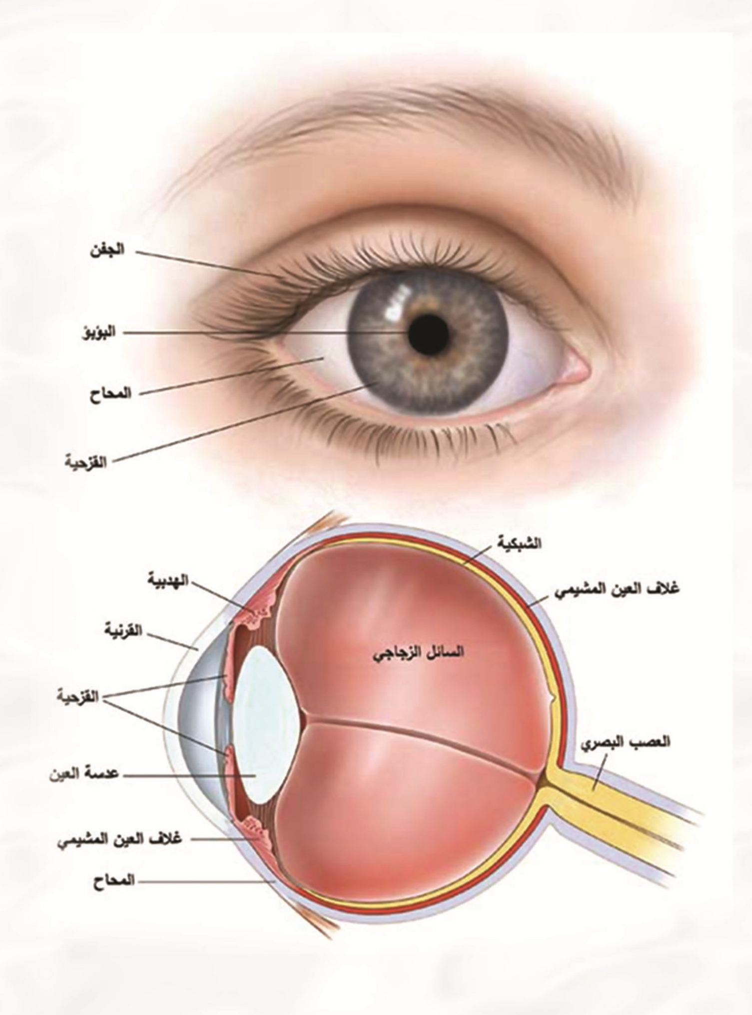
أمراض العين

Ocular Diseases

Aseel O. Hamadeh





الدار العثمانية
OSMANYBOOK

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Contents

الاهداء

إلى كل من ساعدني في نشر هذا الكتاب ، إلى عائلتي وخصوصا أبي وأمي،

إلى أصدقائي وزملائي في العمل، وأيضا إلى طلابي أهدي هذا الكتاب

For all whom helped me to puplish this book, to my family especially my father and my mother, for my friends and my work team, also for my students I gift this book.

Chapter One
History & examination

History and examination

History:

An accurate history and examination are essential for correct diagnosis and treatment.

The failure to take a history and perform a simple examination can lead to conditions being missed that pose a threat to sight, or even to life.

The rate of onset of visual symptoms gives an indication of the cause. E.g. a sudden deterioration in vision tends to be vascular in origin, whereas a gradual onset suggests a cause such as cataract, the loss of visual field may be characteristic, such as the central field loss of macular degeneration.

Difficulties with work, reading, watching T.V, and managing in the house should be identified.

The patient should also be asked exactly what is worrying them, as visual symptoms often cause great anxiety. Appropriate reassurance then can be given.

Questions about particular symptoms:

Some specific questions are important in certain circumstances. E.g a history of ocular trauma or any high velocity injury. Other questions, about the type of discharge in patient with a red eye.

1. Previous ocular history:

1. The patient's red eye may be associated with complications of contact lens wear.
2. A history of server shortsightedness (myopia) considerably increases the risk of retinal detachment.
3. A history of longsightedness (hypermetropia) and typically the use of reading glasses before the age of 40 increases the risk of angle closure glaucoma.
4. Eye drops and eye operations.

2. Medical history:

The medical history may give clues to the cause of the problem.

3. Family history:

As primary open angle glaucoma, because this may be asymptomatic until severe visual damage has occurred. The risk of the disease may be as high as 1 in 10 in first degree relatives, and the disease may be arrested if treated at an early stage. For any disease that has a genetic component, the age of onset and the severity of disease in affected family members can be very useful information.

4. Drug history:

Many drugs affect the eye, and they should always be considered as a cause of ocular problems. E.g steroid drugs in many different forms, many leads to steroid induced glaucoma.

Examination of the visual system:

Vision:

An assessment of visual acuity measures the function of the eye and gives some idea of the patient's disability.

1. Visual acuity is checked with a standard Snellen chart at 6 m. if the room is not large enough, a mirror can be used with a reversed Snellen chart at 3 m.
2. If the top line cannot be discerned, the test can be done closer to the chart.
3. If the chart cannot be read at 1 m, patients may be asked to count fingers.
4. If they cannot do that, to detect hand movements.
5. It may be that they can perceive only light.

Reading vision can be tested with a standard reading type book or, if this is not available, various sizes of newspaper print. There may be quite a difference in the near and distance vision. In propiobia distance vision may be 6/6 without glasses, but the patient may be able to read only larger newspaper print.

Colour vision can be tested by using Ishihara colour plates, witch may give useful information in cases of inherited and acquired abnormalities of colour vision. The ability to detect relative degrees of contrast sensitivity is also important and can be assessed with a Pelli-Robson chart because some eye problems (such as cataract) may cause a significant reduction in contrast sensitivity, despite good Sellen visual acuity.

Field of vision:

Test of visual field may give clues to the site of any lesion and the diagnosis. It is important to test the visual field in any patient with unexplained visual loss.

1. *Location of the lesion*—Unilateral field loss in the lower nasal field suggests an upper temporal retinal lesion. Central field loss usually indicates macular or optic nerve problems.
2. *Diagnoses*—A bitemporal field defect is most commonly caused by a pituitary tumour.

To test the visual field—

1. The patient should be seated directly opposite the examiner.
2. Should be asked to cover the eye that is not being tested and to look at the examiner's face.
3. Testing the visual field with peripheral finger movements will show severe defects, but a more sensitive test is the detection of red colour, because the ability to detect red tends to be affected earlier. A red pin is moved in from the periphery and the patient is asked when they can see something red.

In case of gross defect, the patient will not be able to see part of the examiner's face and may be able to indicate this precisely: "I can't see the centre of your face".

The pupils:

A bright torch is essential. A pupil stuck down to the lens is a result of inflammation within the eye. A peaked pupil after ocular injury suggests perforation with the iris trapped in the wound. A vertically oval unreactive pupil may be seen in acute closed angle glaucoma.

The pupil's reaction to a good light source is a simple way of checking the integrity of the visual pathways. When testing the direct and consensual pupil reactions to light, the illumination in the room should be reduced and the patient should focus in a distant point. By the time pupils do not react to direct light, the damage is very severe. A much more sensitive test is the relative difference in pupillary reactions. Move the torchlight to and fro between the eyes, not allowing time for the pupils to dilate fully.

1. If one of the pupils continues to dilate when the light shines on it, there is a defect in the visual pathway on that side (relative afferent pupillary defect) RAP. Neurological disease must be suspected.
2. Other important and potentially life threatening conditions in which the pupils are affected include Horner's syndrome, where the pupil is small but reactive with an associated ptosis.
3. The will known Argyll Robertson pupils caused by syphilis (bilateral small irregular pupils with light-near dissociation) are rare.
4. In a third nerve palsy there is ptosis and the eye is divergent. The pupil size and reactions in such a case give important clues to the aetiology. If the pupil is unaffected, the cause is likely to be medical. If the pupil is dilated and fixed, the cause is probably surgical.
5. Any different in the colour of the two irides (heterochromia iridis) should be noted as this may indicate congenital Horner's syndrome, certain ocular inflammatory conditions (Fuch's heterochromic cyclitis), or an intraocular foreign body.

Eye position and movement:

The appearance of the eyes shows the presence of any large degree of misalignment. The position of the corneal reflections help to confirm whether there is a true "squint".

Patients should be asked if they have any double vision. If so, they should be asked to say whether diplopia occurs in any particular direction of gaze. It is important to exclude palsies of the third or sixth cranial nerves, as there may be secondary to life threatening conditions. Complex abnormalities of eye movements should lead you

to suspect myasthenia gravis or dysthyroid eye disease. The presence of nystagmus should be noted.

A protruding globe (proptosis) or sunken globe (enophthalmos) should be recorded.

Eyelids, conjunctive, sclera, and cornea:

Should be performed in good light and with magnification. You will need:

- an ophthalmoscope with a blue filter for use with fluorescein.
- a magnifying aid.

The lower led should be gently pulled down to show the conjunctival lining and any secretions in the lower fornix.

The anterior chamber should be examined, looking specifically at the depth and for the presence of pus or blood.

If there are symptoms of "grittiness", a red eye or any history of foreign body, the upper eyelid should be everted. This should not be done, however, if there is any question of ocular perforation, as the ocular contents may prolapse.

Conjunctive and sclera—Look for location or generalized inflammation and pull down the lower lid and evert upper lid.

Cornea—Look at clarity and stain with fluorescein.

Anterior chamber—Check for blood and pus; also check chamber depth.

The drainage angle of the eye can be checked with a special lens (gonioscope).

Ophthalmoscopy:

A direct ophthalmoscope can be used to allow intraocular structures to be seen. Specific contact and non-contact lenses are used during the examination, and the ophthalmologist should use a slit-lamp microscope or head-mounted ophthalmoscope.

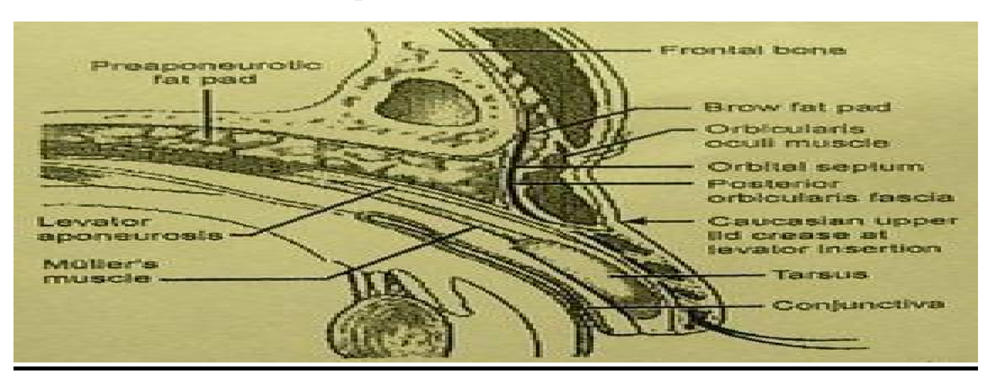
To get a good view, the pupil should be dilated. The best dilating drop is tropicamide 1%, which is short acting and has little effect on accommodation.

Using ophthalmoscope:

1. The direct ophthalmoscope should be set on the “0” lens.
2. The patient should be asked to fix their gaze on an object in the distance.
3. The examiner should use his right eye to examine the patient’s right eye, and vice versa.
4. The light should be shone at the eye until the red reflex is elicited (this red reflex is the reflection from the fundus and is best assessed from a distance of about 50 cm). if the red reflex is either absent or diminished, this indicates an opacity between the cornea and retina.
5. The optic disc should then be located and brought into focus with the lenses in the ophthalmoscope.
6. The retina should be scanned for abnormalities such as haemorrhages, exudates, or new vessels.
7. The green filter on the ophthalmoscope helps to enhance blood vessels and microaneurysms.
8. Finally the macula should be examined for the pigmentary changes of age-related macular degeneration and the exudates of diabetic maculopathy.

Chapter Two
Eye lid disorders

Eye lid disorders



Applied anatomy:

1. The grey line:

Divides the eyelids into an anterior lamella (composed of skin and orbicularis) and posterior lamella (composed of tarsal plate and conjunctiva)

2. Glands:

a. Meibomian glands:

Secret the outer lipid layer of the tear film

b. Glands of zeis:

Sebaceous glands that are associated with lash follicles

c. Glands of moll:

Are sweat glands

3. The lashes:

Are more numerous in the upper than the lower lid

4. Upper lid elevators:

a. The levator aponeurosis

b. Muller muscle

5. Lower lid retractor:

a. The inferior tarsal aponeurosis

b. The inferior tarsal muscle

6. Lymphatic drainage:

The upper lid and lateral canthus drain into the preauricular nodes. Where as the lower lid and medial canthus drain into the submandibular nodes.

Malpositions of the eyelids and eyelashes:

Trichiasis

- Is an inward turning of eye lashes which causes annoying irritation which is made worse by blinking.
- Is a common acquired unilateral or bilateral condition.



• *Causes:*

- isolated
- Associated with scarring of the lid margin such as that caused by chronic Plepharitis and trachoma.

• *Signs:*

- Posterior misdirection of lashes arising from their normal sites of origin.
- Trauma to the corneal epithelium.

• *Treatment:*

1. ***Epilation***: with forceps is simple and effective but recurrence within a few weeks.
2. ***Electrolysis***: is useful for a few isolated lashes.
3. ***Cryotherapy:*** is very effective in eliminating many lashes simultaneously.
4. ***laser ablation:*** is useful when only a few scattered lashes require treatment.

Ectropion

Is outward turning of lid margin and sagging eyelid that leaves the eye exposed and dry.



• *Classifications:*

1. anatomical:
   - punctual
   - medial
   - lateral
   - tarsal (complete)
2. according to time of onset:
   - congenital
   - acquired:
     - involution
     - cicatricial
     - paralytic
     - mechanical

• *Signs and symptoms:*

1. corneal exposure: excessive tearing
2. red, irritated eyelid, burning
3. gritty, sandy feeling
4. Keratinization of the palpebral conjunctiva.
5. visual loss

• Complications:

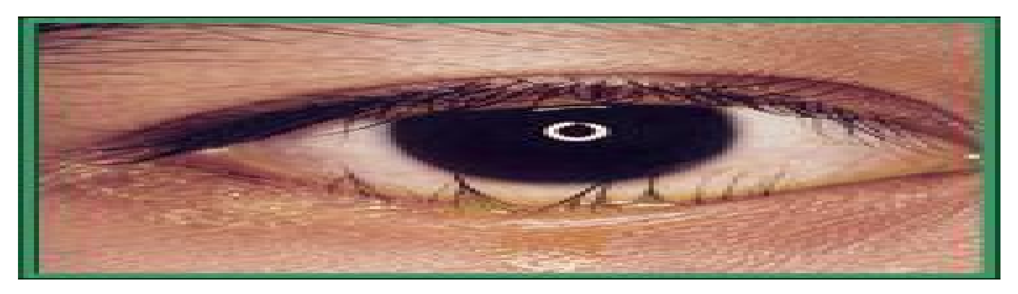
1. eye infections
2. corneal abrasions
3. corneal ulcer
4. Conjunctival keratinization
5. Epiphora and pain
6. punctual stenosis

• Treatment:

1. Medical: antibiotics, artificial tears.
2. surgical

Entropion

Is an inward turning of lid margin



• Types of entropion:

1. involutional
2. cicatricial: scarring of the conjunctiva
3. congenital
4. acute spastic

• Signs and symptoms:

1. excessive tearing
2. eye irritation
3. redness
4. eye discomfort
5. decrease vision if the cornea is damaged

• ***Complications:***

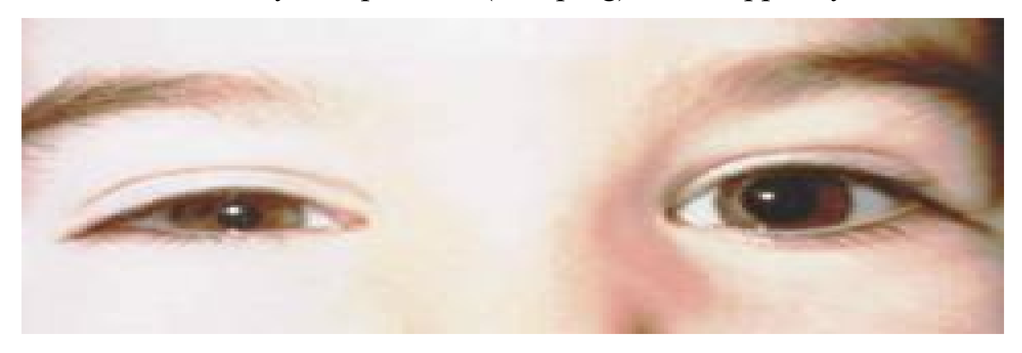
1. Corneal breakdown
2. ulcer formation
3. Epiphora
4. Pain

• ***Treatment:***

1. Medical: lubricating eye drops and ointment, plucking the offending eyelashes.
2. surgical

Ptosis

Is an abnormally low position (drooping) of the upper eyelid.



• ***Types of ptosis:***

1. Congenital
2. Acquired

• ***Symptoms:***

1. drooping of the upper eyelid in one or both eyes
2. undesirable facial appearance due to drooping of the upper eyelid
3. elevated chin in sever ptosis
4. stiff nick due to constant chin elevation
5. eye fatigue from straining to keep eye open
6. decreased vision when the droop is severe and covers the pupil

• Causes of ptosis:

1. myogenic: The skeletal muscle fibers of the LPS are replaced by fibroadipose tissue
2. neurogenic: caused by innervational defect
3. mechanical: mass defect
4. aponeurotic: due to failure of the levator aponeurosis to insert at its normal position on the anterior tarsus
5. traumatic: caused by direct trauma

• Indication of treatment of ptosis:

1. cosmetic
2. visual

• Treatment:

1. involutional: surgery
2. myogenic and neurogenic: systemic treatment of possibly life threatening problems
3. mechanical: treat underlying cause

Allergic disorders:

Chronic marginal Blepharitis



Classification:

1. Anterior:
   - Staphylococcal
   - Seborrhoeic
   - Mixed
2. Posterior:
   - Meibomian seborrhea
   - Meibomianitis
3. Mixed anterior and posterior

Anterior Blepharitis:



• **Symptoms:**

1. Burning
2. Grittiness
3. Mild photophopia
4. Crusting and redness of lid margins

   These symptoms are usually worse in the morning

• **Signs:**

a. Staphylococcal Blepharitis:

   1. Hyperaemia of anterior lid margin
   2. Telangiectasia of anterior lid margin
   3. Hard scales mainly located around the bases of the lashes

b. Seborrhoeic Blepharitis:

1. Hyperaemic and greasy anterior lid margin
2. Sticking together of lashes
3. Soft scales and located anywhere on the lid margin and lashes

c. Sever long standing anterior Blepharitis (staphylococcal):

1. Hypertrophy
2. Scarring of lid margin
3. Madarosis
4. Trichiasis
5. Poliosis

• **Associations:**

1. External hordeola (stye)
2. Tear film instability is present in 30-50 % of cases
3. Papillary conjunctivitis (result from hypersensitivity to staphylococcal exotoxins)

• **Treatment:**

1. Lid hygiene:

Using lid scrub, or a cotton bud dipped in a solution of baby shampoo or a weak solution of bicarbonate

2. Antibiotic ointment:

Such as fucidin or chloramphenicol (used for acute folliculitis)

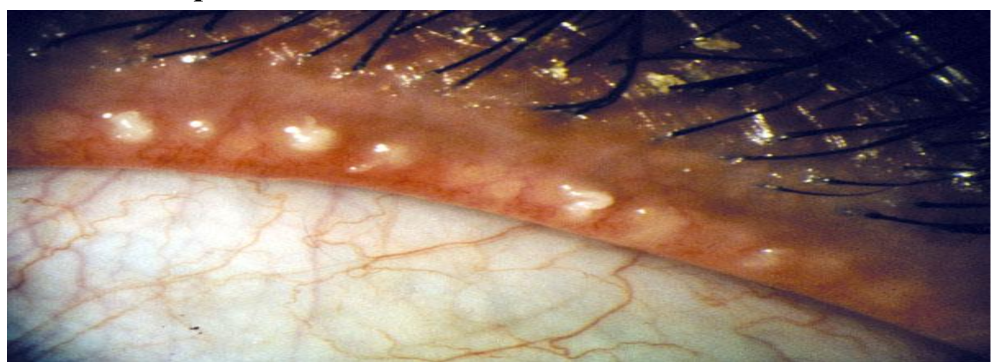
3. Weak topical steroids:

Such as fluorometholone administered short term (useful for secondary papillary conjunctivitis and marginal Keratitis)

4. Tear substitutes:

Required for associated tear film instability

Posterior Blepharitis:



• **Signs and symptoms:**

a. Meibomian seborrhea:

1. Excessive meibomian gland secretion
2. The meibomian glands orifices are capped by small oil globules

   Pressure on the tarsus result in expression of copious amounts of meibomian oil.
3. The tear film is oily and foamy

b. Meibomianitis:

1. Inflammation of the meibomian glands
2. Obstruction of the meibomian glands
3. The posterior lid margin shows hyperaemia
4. Telangiectasia and obstruction of meibomian gland orifices
5. Long standing cases: cyst dilatation of meibomian ducts, with thickening and notching of the lid margin
6. Expressed meibomian gland secretion

• **Complications:**

1. Chalazion formation
2. Tear film instability in about 30% of patients
3. Papillary conjunctivitis and inferior corneal epithelial erosions

• **Treatment:**

1. Systemic tetracyclines

2. Erythromycin

3. Others:

a. Lid hygiene

b. Worm compresses

c. Topical sodium fusidate gel

Benign nodules and cysts



Chalazion (meibomian cysts)



Is a chronic, sterile, lipogranulomatous inflammatory lesion caused by blockage of meibomian gland orifices and stagnation of sebaceous secretions

• ***Risk factors:***

1. Acne rosacea

2. Seborrhoeic dermatitis

• ***Clinical features:***

Is at any age with a gradually enlarging painless nodule.

An upper lid Chalazion may press on the cornea induce astigmatism and cause blurred vision

• *Signs:*

A non-tender, roundish, firm lesion within the tarsal plate of variable size which may be multiple or bilateral.

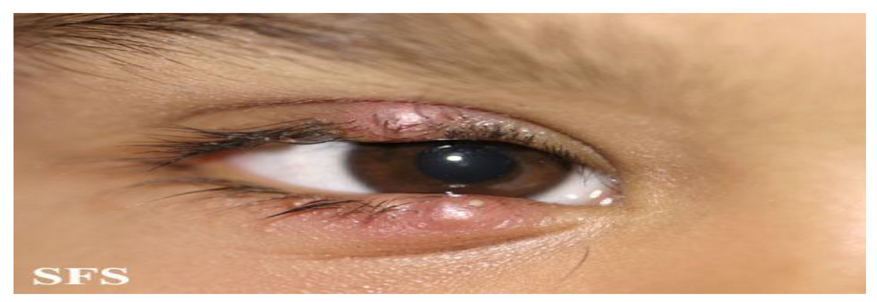
Eversion of the lid may show an associated polypoidal granuloma if the lesion has ruptured through the tarsal conjunctiva.

• *Treatment:*

1. Surgery
2. Steroid injection
3. Systemic tetracycline



Hordeolum



• *Classification:*

1. Internal hordeolum
2. External hordeolum

Internal hordeolum:

Is an abscess caused by an acute staphylococcal infection of meibomian gland.

• Signs:

A tender, painful swelling within the tarsal plate.

The lesion may enlarge and then discharge either posteriorly through the conjunctiva or anteriorly through the skin.

• Treatment:

Incision and curettage may be required if a residual nodule remains after the acute infection has subsided.

External hordeolum:

Is an acute staphylococcal abscess of a lash follicle and it is associated gland of zeis or moll which usually affects children.

- **Signs:**

A tender swelling in the lid margin pointing anteriorly through the skin.

Multiple lesions may be present and occasionally minute abscesses may involve the entire lid margin.

- **Treatment:**

Hot compresses and epilation of the lash associated with the infected follicle may hasten resolution.

Stye

Is an infection of the subaceous glands of zeis at the base of the eyelashes, or an infection of apocrine sweat glands of moll.



- **Causes:**

1. a Staphylococcus aureus bacterial infection
2. blocking of the oil gland at the base of the eyelashes

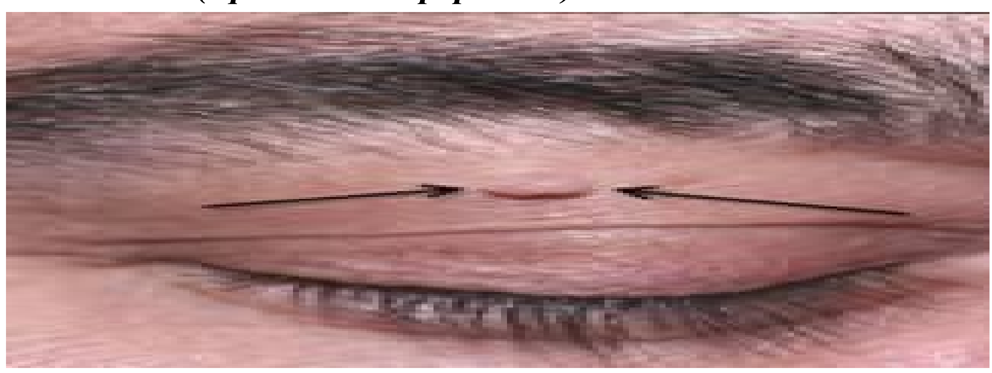
- **treatment:**

1. hygiene
2. warm compresses to help it to discharge
3. chloramphenicol ointment should be used

Stye will last from up to 3 weeks to 2 yrs if not treated, and only 1 week if treated properly.

Benign tumours:

1. Viral wart (squamous cell papilloma):



Is the most common benign tumour of the eyelids which is usually found in adults.

- Signs:

A pedunculated or broad-based (sessile) lesion with a characteristic raspberry-like surface.

- Treatment:

Excision or laser ablation.

2. Seborrhoeic keratosis (basal cell papilloma):



Is a common slow-growing condition found on the face and eyelids of elderly individuals.

- Signs:

A discrete, greasy, brown, flat, round or oval lesion with a friable verrucous surface and a “ stuck-on” appearance.

Occasionally the lesion may be pedunculate.

- Treatment:

Curettage of small flat lesions and excision of pedunculated lesions.

3. Actinic (solar) keratosis:



Is the most common pre-malignant skin lesion but rarely develops on the eyelids.

It typically affects elderly, fair-skinned individuals who have been exposed to excessive sunlight and most frequently occurs on the forehead and backs of the hands.

- Signs:

A flat, scaly, hyperkeratotic lesion.

Occasionally the lesion is nodular or wart-like and may be associated with a cutaneous horn.

- Treatment:

Biopsy to confirm the diagnosis, followed by either excision or cryotherapy.

4. Cutaneous horn:



Is an uncommon lesion which is occasionally associated with underlying actinic keratosis or squamous cell carcinoma.

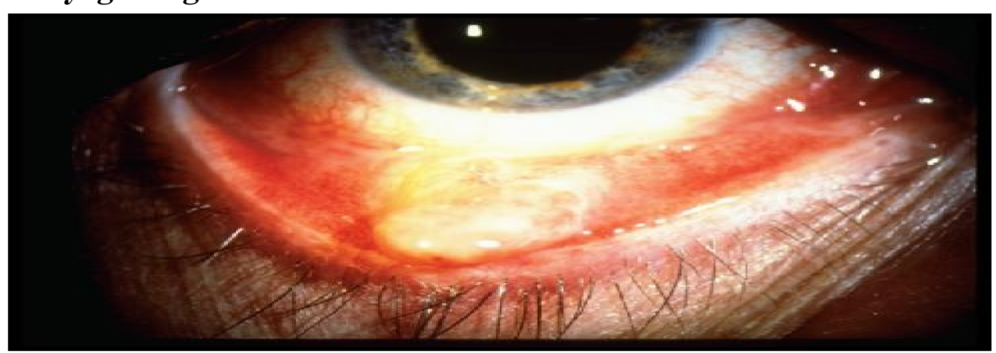
- Signs:

A hyperkeratotic lesion protruding through the skin.

- Treatment:

Biopsy to determine the underlying pathology, followed by excision.

5. Pyogenic granuloma:



Is a fast-growing vascularized proliferation of granulomatous tissue which is usually antedated by surgery, trauma or infection, although some cases are idiopathic.

- Signs:

A pinkish, pedunculated or sessile mass which may bleed following relatively trivial trauma.

- Treatment:

Excision

6. Melanocytic naevus:



Is composed of a typical melanocytes (naevus cells).

The clinical appearance, classification and potential for malignant transformation of naevi are determined by their histological location within the skin as follows:

a. Intradermal naevus:

The most common, is usually elevated and frequently has a papillomatous configuration.

It may be non-pigmented or brown-black in colour.

When located on the eyelid margin, lashes may grow through the lesion.

Occasionally symmetrical lesions may be present on adjacent portions of the upper and lower lids (kissing naevi).

Histologically the naevus cells are confined to the dermis and have no malignant potential.

b. Junctional naevus:

Is usually flat, well circumscribed and uniformly brown in colour.

The naevus cells are located at the junction of the epidermis and dermis and have a low potential for malignant transformation.

c. Compound naevus:

Has both intradermal and junctional components and is usually brown.

It has a low potential which is related to the junctional component.

7. Keratocanthoma:



Is an uncommon, benign but rapidly growing tumour which usually occurs in otherwise healthy fair-skinned individuals with a history of chronic sun exposure.

It is found more frequently than would be expected by chance in patients on immunosuppressive therapy following renal transplants.

Keratocanthoma may have a clinically similar appearance to squamous cell carcinoma.

- Signs:

1. A pink papule which may double or treble in size within a few days.
2. The lesion generally stops growing and remains static for 2-3 months, after which time it starts to involute spontaneously.
3. At the end of the growth phase the lesion is a firm, dome-shaped nodule.
4. During the period of regression the central part of the lesion becomes hyperkeralotic and a keratin-filled crater may develop.
5. Complete involution may take up to a year and usually leaves a residual ugly scar.

- Treatment:

Excision, especially if there is no sign of spontaneous involution.

8. Strawberry naevus (capillary haemangioma):

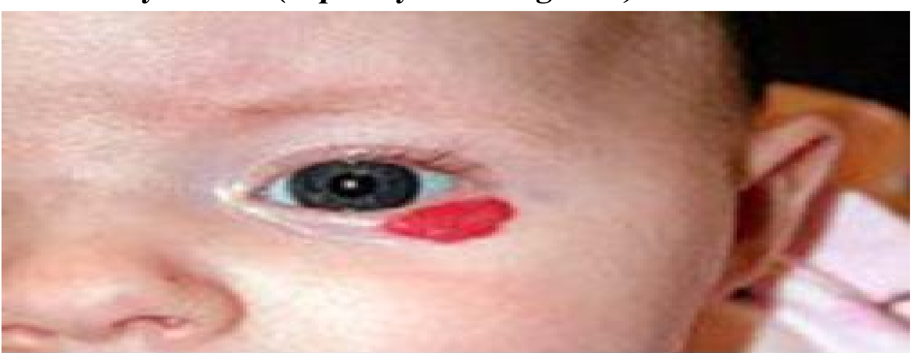


Fig. 1 A capillary hemangioma is an abnormal overgrowth of blood vessels that is sometimes referred to as a "strawberry" birthmark.

Is one of the most common tumours of infancy and presents shortly after birth.

The female to male ratio is 3:1 and occasionally the tumour is familial.

- Signs:

1. A unilateral, raised, red lesion which blanches with pressure and may swell on crying.

   A large tumour on the upper lid may cause mechanical ptosis.

2. Usually grow quickly during the first year of life and stops during the second year.

3. At about the age of 2 years the tumour begins to involute spontaneously with complete resolution occurring in 40% of patients by the age of 4 years and 70 % by the age of 7.

- Systemic associations:

1. Kasaback-merritt syndrome
2. Maffuci syndrome

- Treatment:

1. Laser: to close blood vessels in early lesions
2. Injection of steroid: is the most frequently used method
3. Systemic steroid: for extensive lesions

4. Subcutaneous injection of interferon alpha-2b: for the treatment of steroid-resistant
5. Surgical resection: in selected cases

9. Part-wine stain (naevus flammeus):



Is a rare congenital, subcutaneous cavernous haemangioma which most frequently occurs on the face.

The lesion is usually unilateral and segmental but may be bilateral.

- Signs:

1. A sharply demarcated, soft, pink patch which does not blanch with pressure.
2. With age, the lesion does not grow but darkens to red or purple.
3. The overlying skin may become hypertrophied, coarse, nodular and friable and may bleed or become infected.

- Treatment:

Erbium laser

Malignant tumours:

1. Basal cell carcinoma (BCC):



Is the most common human malignancy, which most frequently affect the elderly patients.

- Risk factors:

1. Fair skin
2. Inability to tan
3. Chronic exposure to sunlight

The tumour is slow growing and locally invasive but non-metastasizing.

- Clinical types:

1. Nodular-ulcerative:

BCC starts as a shiny, firm, pearly nodule with small dilated blood vessels on its surface.

Initially the growth is slow and may take 1-2 years to reach a diameter of 0.5 cm. if the tumour is not recognized and treated at an early stage, subsequent growth is faster and the lesion develops central ulceration, raised rolled edges with dilated blood vessels over its lateral margins.

With time it may erode a large portion of the eyelid.

2. Sclerosing:

Is less common and may be difficult to diagnose.

The margins of the tumour may be impossible to delineate clinically and the lesion tends to be much more extensive on palpation than inspection.

On cursory examination a sclerosing BCC may simulate a localized area of unilateral "chronic Blepharitis".

2. Squamous cell carcinoma (SCC):



Is much less common, but potentially a more aggressive tumour than BCC, with eventual metastasis to regional lymph nodes.

There may also be perineural spread to the intracranial cavity via the orbit.

Immunocompromised patients with AIDS or following renal transplants are at increased risk.

It occurs most commonly in elderly individuals with a fair complexion and a history of chronic sun exposure and skin damage.

The diagnosis of SCC may be difficult because other malignant tumours, precancerous lesions and benign tumours may mimic SCC.

Clinically, SCC may be indistinguishable from BCC but it does not usually manifest surface vascularization and grows more rapidly.

- Clinical types:

1. *Plaque-like SCC:*

   Is characterized by a roughened, scaly, erythematous, hyperkeratotic plaque which may arise at the site of pre-existing actinic keratosis.

2. *Nodular SCC:*

   Is characterized by a hyperkeratotic nodule which may develop crusting erosions and fissures.

3. *Ulcerating SCC:*

   Has a red base and sharply defined, indurated and everted borders.

3. Sebaceous gland carcinoma (SGC):

Is a very rare slow growing tumour which most frequently affects the elderly.

It most frequently arises from the meibomian glands, although on occasion it may arise from the glands of zeis or sebaceous glands.

In contrast to BCC and SCC, the tumour occurs more commonly on the upper eyelid, where meibomian glands are more numerous.

The clinically diagnosis of SGC is frequently difficult because, in its early stages, external signs of malignancy may be sutable so that the tumour may resemble a less aggressive lesion.

Bad prognostic features are upper lid involvement, tumour size of 10 mm or more and a duration of symptoms of over 6 months.

- Clinical types:

1. Nodular meibomian gland carcinoma:

Presents with a discrete, hard nodule most commonly within the upper tarsal plate and may masquerade as a “Chalazion”.

It is there recommended that any Chalazion of an unusual consistency should undergo full-thickness resection and histological examination.

Unless treated, the nodule may become very large.

2. Spreading meibomian gland carcinoma:

Infiltrates into the dermis and causes a diffuse thickening of the lid margin, and may also invade the conjunctiva.

Pagetoid spread refers to extension of the tumour within the epithelium of the palpebral, forniceal or bulbar conjunctiva.

This may lead to mistaken diagnosis of an inflammatory condition such as “chronic conjunctivitis”, “superior limbic keratoconjunctivitis” or “cicatrecial pemphigoid”.

3. Gland of zeis carcinoma:

Is very rare, is characterized by a discrete, slow-growing, nodular or ulcerative lesion on the lid margin.

Blepharospasm

Is forceful, uncontrollable closing of the eyelids. Often it is affect both eyes but it can affect one eye. As the condition worsens, the blinking or winking occurs more often. The eyelids can be closed for longer than a normal blinking reflex, which may cause vision problems.

Symptoms:

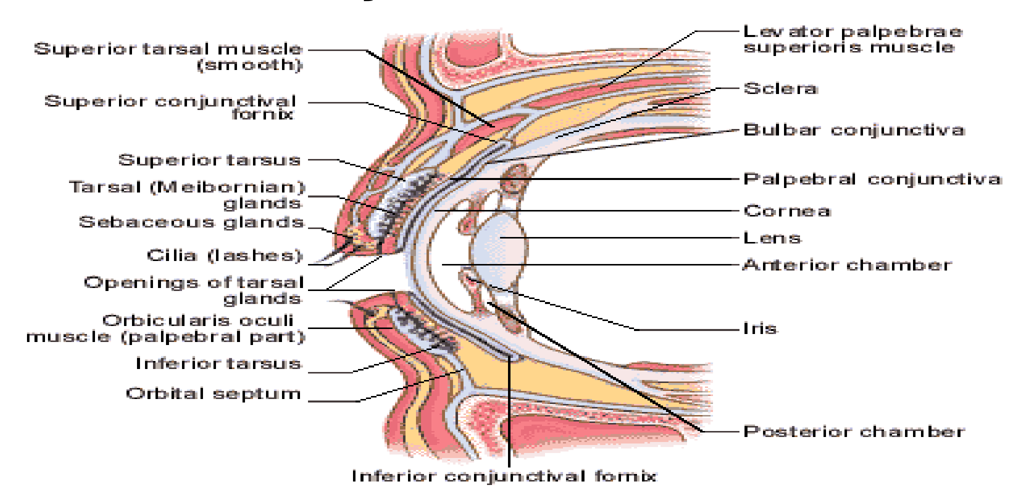
1. Winking, blinking, or squinting that you can't control
2. Trouble keeping your eyes open
3. Sensitivity to light.

Treatment:

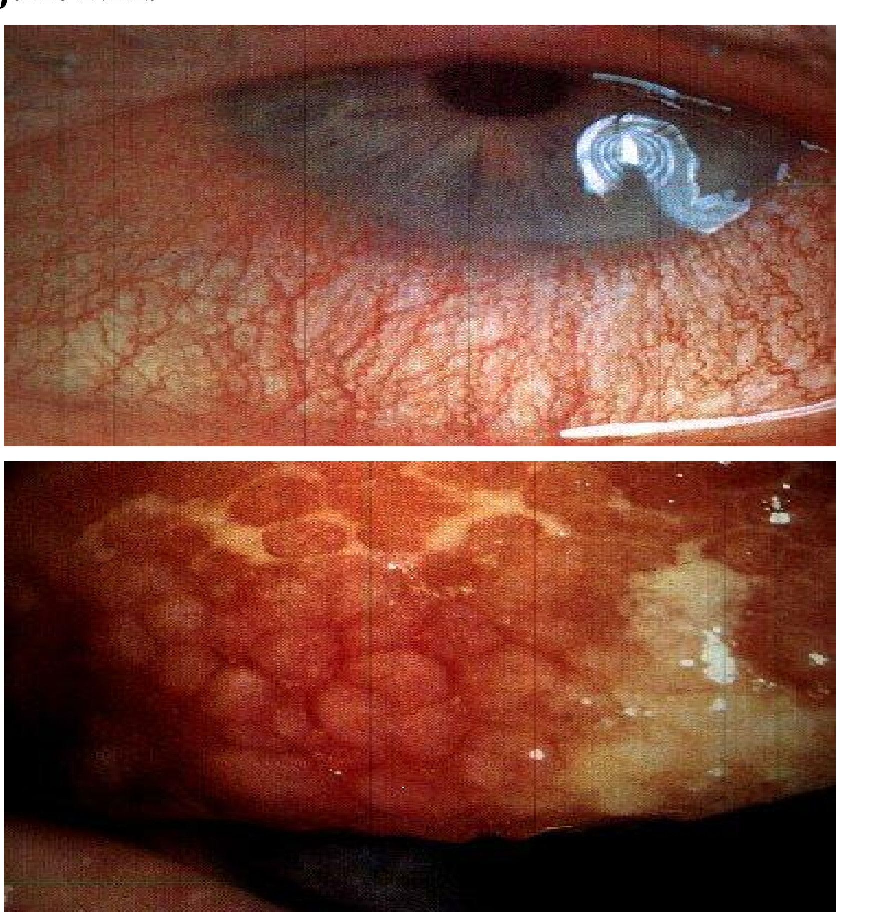
1. Injection of a medicine (botox)
2. Medicines taken by mouth
3. Surgery
4. Stress management.

Chapter Three
Conjunctival disorders

Conjunctival disorders



Conjunctivitis



Bacterial conjunctivitis:

Clinical features:

1. Palpebral redness
2. Purulent discharge
3. Papillae
4. Edema of eyelids
5. Ocular irritation

Common pathogens:

1. Staphylococcus
2. streptococcus
3. pneumococcus
4. haemophilus
5. pseudomonas

Treatment:

1. culture
2. warm compresses
3. clean lids of discharge
4. topical antibiotics.

Viral conjunctivitis:

Symptoms:

1. itching
2. burning
3. palpebral redness

Signs:

1. watery discharge
2. Conjunctival follicles
3. Enlarged pre-auricular lymph nodes.

4. may be also lid oedema.

5. excessive lacrimation

Treatment:

Viral conjunctivitis is generally a self limiting condition.

- antibiotic eye drops provide symptomatic relief and help prevent secondary bacterial infection
- hygiene the period of infection is often longer than with bacterial pathogens
- steroid eye drops may be necessary for some patients with chronic infection .

Trachoma:



- This is the commonest infective cause of blindness in the world.
- The housefly acts as a vector and the disease is encouraged by poor hygiene and overcrowding in a dry, hot climates
- The hallmark of the disease is subconjunctival fibrosis caused by frequent re-infections associated with the unhygienic condition.
- Blindness may occur due to corneal scarring from recurrent Keratitis and Trichiasis.

Treatment:

- oral or topical tetracycline or erythromycin.
- Entropion and Trichiasis require surgical correction.

Allergic conjunctivitis:

This may be divided into acute and chronic forms:

1. acute (hayfever conjunctivitis)

   Signs and symptoms:

   - itchiness
   - conjunctival swelling
   - lacrimation

2. vernal conjunctivitis (spring catarrh)

   Signs and symptoms:

   - itchiness
   - photophobia
   - lacrimation
   - papillary conjunctivitis on the upper tarsal plate
   - Limbal follicles and white spots.
   - Punctuate lesions on the corneal epithelium

Treatment:

- Antihistamines and mast cell stabilizers.
- Topical steroids in sever cases.

Note:

Contact lens wearers may develop an allergic reaction to their lenses or to lens cleaning materials leading to a ***giant papillary conjunctivitis (GPC)*** with a mucoid discharge.

This may response to topical treatment with mast cell stabilizers.

It is often necessary to stop lens wear for a period or even permanently.

Conjunctival degeneration:

- Cysts are common in the conjunctiva, they rarely cause symptoms but it necessary can be removed.

- Pingueculae and pterygia are found on the interpalpebral bulbar conjunctiva, they are thought to result from excessive exposure to the reflected or direct UV component of sunlight.

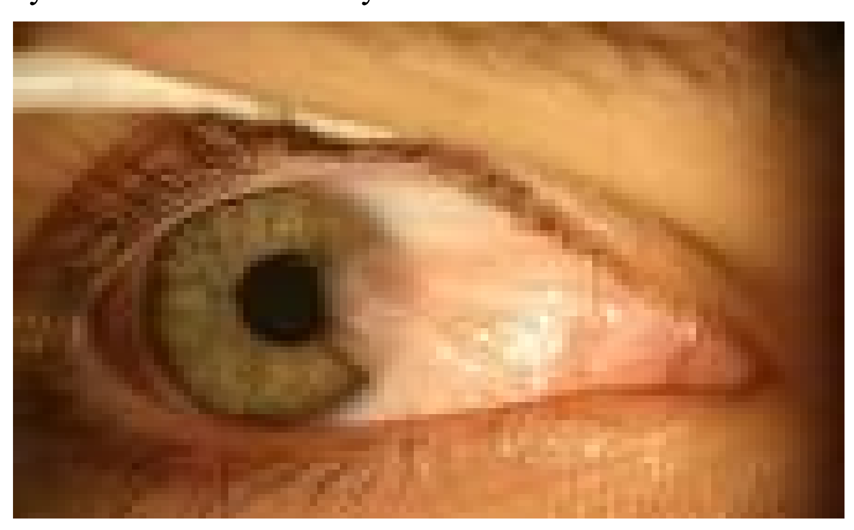
Histologically the collagen structure is altered.

• Pingueculae are yellowish lesions that never impinge on the cornea.

• Pterygia are wing shaped and located nasally, which the apex towards the cornea onto which they progressively extend.

They may cause irritation and, if extensive, may encroach onto the visual axis.

They can be excised but may recur.



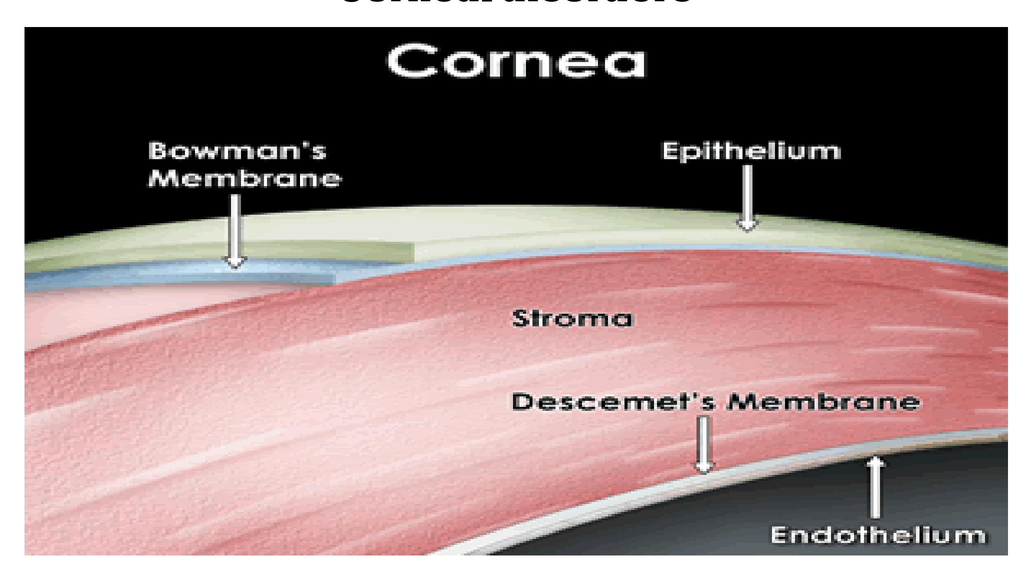
Conjunctival tumours:

Rare but include:

- Squamous cell carcinoma.
- Malignant melanoma

Chapter Four
Corneal disorders

Corneal disorders



Applied Anatomy:

The average corneal diameter vertically is 11.5 m and horizontally is 12 mm.

Layers of the cornea:

1. The epithelium:

Is stratified, squamous and non-keratinized, and comprises:

a. A single layer of basal columnar cells.

b. Two or three rouls of wing cells.

c. Two layers of squamous surface layers.

2. Bowman layer:

Is the acellular superficial layer of the stroma which scars when damaged.

3. The stroma:

Makes up 90% of corneal thickness.

It is composed of regularly orientated layers of collagen fibrits whose spacing is maintained by proteoglycen ground substrance

(chondroitin sulphate and keratin sulphate) with interspersed modified fibroblasts (keratocytes)

4. Descement membrane:

Composed of a fine lattice work of collagen fibrils.

5. The endothelium:

Consists of a single layer of hexagonal cells.

Microbial Keratitis:

Bacterial Keratitis:

• Predisposing factors:

Bacteria capable of penetrating intact epithelium include " Neisseria gonorrhoeae" and "H. influenza".

Other bacteria are capable of producing Keratitis only after compromisation of epithelial integrity, associated with the following factors:

1. Contact lens wear:

Particularly extended – wear soft lenses.

2. Pre-existing corneal disease:

Such as trauma, bullous keratopathy, diminished corneal sensation.

3. Other factors include:

a. Chronic blepharoconjunctivitis.

b. Chronic dacryocystitis.

c. Tear film deficiency.

d. Topical steroid therapy.

e. Hypovitaminosis.

• Clinical features:

Presentation is with foreign body sensation which progresses to photophopia. Blurring of vision, pain, eyelid oedema and discharge.

In contact lens-wearers symptoms maybe masked or delayed.

• **Signs:**

1. Conjunctinal and circumcorneal injection.
2. An epithelial defect associated with an infiltrate around the margin and base.
3. Enlargement of infiltrate associated with stromal oedema.
4. Secondary sterile anterror uveitis with hypopyon.
5. Progressive ulceration may lead to corneal perforation and bacterial endophthalmitis.

• **Treatment:**

1. Initial treatment should be with broad-spectrum topical antibiotics because polymicrobial infections are common and an unproductive gram stain doesn't exclude bacterial infection.
2. Dual therapy: involves a combination of two fortified antibiotics to cover common gram-positive and gram-negative pathogens, in the form of an aminoglycoside and a cephalosporin.
3. Monotherapy: With a fluroquinolone (i.e. ciprofloxacin 0.3% or ofloxacin 0.3%)

Fungal Keratitis:

Although rare, fungal infection (keratomycosis) may have devastating effect.

Fungs can cause sever stromal necrosis and enter the anterior chamber by penetrating an intact descement membrane. Once it enters the anterior chamber, the infection becomes very difficult to control in part due to poor penetration of antimycotic agents.

The most common pathogens are:

1. Flamentous fungi.
2. Candida albicans.

Presentations:

1. Gradual onset of foreign body sensation.
2. Photophopia.
3. Blurred vision.
4. Discharge.

• **Signs:**

a. Filamentous Keratitis:

1. A grayish, stromal infiltrate with adry texture and indistinct mangins.
2. Surrounding, satellite, feathery, finger-like leasions and immune ring infiltrates.
3. An underlying endothelial plaque and hypopyon may be present.

b. Candida Keratitis:

Characterized by a yellow-white ulcer associated with dense suppuration similar to a bacterial Keratitis.

• **Treatment:**

Before instituting antimycotic therapy, corneal scraping with a surgical blade should be performed in order to reduce the fungal load and enhance penetration of antifungal agents.

1. Topical treatment should be for 6 weeks:
   a. Filamentous infection:
      ▪ Natamycin 5%.
      ▪ Amphotericin 0.15% may be added as a second agent if necessary.
   b. Candida infection:
      ▪ Imidazole 1% or flucytosine 1%.
      1. Systematic antimycotics:
         May be required for sever Keratitis or endophthalmitis.
      2. Therapeutic penetrating keratoplasty:
         May be required in unresponsive cases.

Acanthamoeba Keratitis:

Acanthamoeba are found in air, soil and fresh or brackish water.

• **Presentations:**

1. Blurred vision.
2. Sever pain.

• **Signs:**

1. Limbitis.
2. The overlying epithelium may be intact or pseudo-dentritic Keratitis.
3. Gradual enlargement and coalescence of the infiltrates may form a central or paracentral ring.
4. Small satellite lesion may develop peripheral to the ring.
5. Slowly progressive stromal opacification, scleristis and ultirnately descemetocele formation.

• **Investigation:**

1. Staining.
2. Cultures.

• **Treatment:**

1. Topical amoebicides.
2. Topical steroids.
3. Therapeutic penetrating keratoplasty.

Luetic interstitial Keratitis (IK):

Is an inflammation of the corneal stroma without primary involvement of the epithelium or endothelium.

• **Presentation:**

Occun between 5 – 25 years with a cute bilateral pain and sever blurring of vision.

• **Signs:**

1. An inflamed raised sector of limbus from which deep vessels invade the corneal stroma. Associated cellular infiltration and corneal clouding obscure the outline of the vessels, resulting in the characteristics “salmon-patch”.
2. Anterior uveitis is present but may be obscured by corneal clouding.

3. After several months the cornea begins to clear and the vessels become non-perfused (ghost vessels).
4. If the cornea later becomes inflamed for any reason, the vessels may refill with blood and rarely bleed.
5. Healing is associated with stromal thining, scarring and often flattening of the cornea.
6. The inactive stage is characterized by a central deep stromal scar of variable density and ghost vessels.

• **Treatment:**

1. Systemic penicillin.
2. Tropic steroids.
3. Cycloplegics.

Microsporidial Keratitis:

Are small, ubiquitous, obligate intracellular, spore-forming protozoa which are opportunistic pathogens that cause multi-system disease primarily in immune compromised patients.

1. Bilateral chronic diffuse punctuate epithelial keratoconjunctivitis:

   May occur in patients with AIDS.

   Treatment is with topical fumagillin and oral albendazole.

   Highly active antiretrovaital therapy (HAART) for AIDS may also be beneficial for the eyes.

2. Unilateral deep stromal Keratitis:

   Affects immunucompetent patients and is very rare.

   There is no effective treatment and most cases require corneal grafting.

Viral Keratitis:

• **Herpes simplex Keratitis:**

▪ **Primary ocular infection:**

This typically occurs in children between the age of 6 months and 5 years, and may be associated with generalized symptoms of a viral illness.

Blepharoconjunctivitis is usually benign, self-limited, and in children may be the only manifestation.

▪ **Signs:**

1. Skin vesicles typically involved the lids and periorbital area.

2. Acute, unilateral, follicular conjunctivitis associated with preauricular lymphadenepathy.

3. Secondary cornalicular abstruction may ensue.

▪ **Treatment:**

Is aimed at preventing Keratitis with aciclovir eye ointment five times a day for 3 weeks.

However, Keratitis is uncommon even without antiviral prophylaxis.

▪ Types of herpex simplex Keratitis:

1. Epithelial Keratitis:

May be at any age with mild discomfort, watering and blurring of vision.

◆**Signs:**

1. Opaque epithelial cells arranged in a coarse punctuate or satellite pattein.
2. Central sensation results in a linear-branching ulcer.
3. Corneal sensation is reduced.
4. Anterior stromal infiltrates subsequently appear under the ulcer, but usually resolve rapidly once the epithelium has healed.

5. Progressive centrifugal en enlargement may result in a large epithelial defect.

6. Following healing. The epithelium may manifest persistent linear branching shapes which represent waves of healing epithelial cells.

◆ **Treatment:**

1. Even without treatment, about 50% of active epithelial lesions heal spontaneously.
2. Acyclovir 3% ointment (zovirax) is used 5 times daily.
3. Ganciclovir 0,15% gel (virgan) is a new preparation which used 5 times daily.
4. Trifluorothymidine 1% drops (F3T) is used every 2 hours during the day.

• ***Disciform Keratitis (endothelitis):***

The exact etiology is controversial.

It present with gradual onset of painless blurred vision which may be associated with haloes around lights.

▪ **Signs:**

1. A central zone of epithelial odema overlying an area of stromal thickening which may be associated with keratic precipitates and folds in descement membrane.
2. A surrounding ring of stromal precipitate may be present, signifying the junction between viral antigen and host antibody.
3. The IOP may be elevated despite only mild anterior uveitis.
4. Old healing lesions are characterized by a faint ring of stromal opacification that permanently marks the border of the previously odematous area.
5. Reduced corneal sensation.

▪ **Treatment:**

1. Initially the steroid and antiviral are given q.i.d.
2. As improvement occurs, the strength of steroid may be reduced and antiviral administered t.i.d.

3. Steroids should be trapered over a period of several weeks, though one drop a day of a week concentrated for a prolonged period may be necessary to prevent rebound.

4. Periodic attempts should be made to taper further or to stop medication altogether.

• ***Stromal necrotic Keratitis:***

Caused by active viral invasion and tissue necrosis.

In rare and may be associated with intact epithelium or many follow epithelial disease.

It present with progressive impairment of vision associated with discomfort and pain.

▪ **Signs:**

1. Cheesy and necrotic stroma reminiscent of a bacterial or fungal infection, or profound interstitial opacification.
2. Associated anterior uveitis with Keratic precipitates underlying the area of active stromal infiltration.
3. If inappropriately treated, scarring, vascularization, lipid keratopathy and even perforation may result.

▪ **Treatment:**

1. Antiviral agents to heal active epithelial disease.
2. A cautious use of steroids and antibiotic cover, may be necessary to retieve symptoms and minimize scarring.

• ***Herpes zoster Keratitis:***

▪ **Acute phase:**

1. ***Acute epithelial Keratitis:*** characterized by small, fine, dendritic or stellate lesions which stain with Fluorescein and rose Bengal.
2. ***Nummular Keratitis:*** characterized by multiple fine granular subepithelial deposits, surrounded by a hole of stromal haze.
3. ***Disciform Keratitis:*** it is usually axial and is almost always preceded by nummular Keratitis.

▪ **Chronic phase:**

1. ***Nummular Keratitis:*** may persist for months, peripheral lesions sometimes forming facets which later become vascularizal and infiltrated by lipid.
2. ***Discomfort Keratitis:*** if neglected, ives rise to scarring, vascularization and lipid deposition.
3. ***Neutrophic Keratitis:*** may lead to sever ulceration, secondary bacterial infection and even perforation.
4. ***Mucous plaque Keratitis:*** characterized by the sudden appearance of elevated mucous plaques which stain britliantly with rose Bengal.

Corneal Dystrophies:

Are a group of progressive, usually bilateral, mostly genetically determined, non-inflammatory, opacifying disorders.

Classifications:

1. Epithelial.
2. Bownon layer.
3. Stromal.
4. Endothelial.

• **Epithelial Dystrophies:**

1. Epithelial basement membrane dystrophy (cogan microcystic or map.dot – fingerprint dystrophy):

▪ **Signs:**

1. Dot-like opacities.
2. Epithelial microcysts.
3. Subepithelial map-like patterns.
4. Whorled fingerprint-like kines.

▪ **Histology:**

Shows thickening of the basement membrane with deposition of fibrillary protein between the basement membrane and Bowman layer.

There is also absence of hemidesmosomes of the basal epithelial cells, which is responsible for recurrent corneal erosions.

▪ **Treatment:**

The same of recurrent corneal erosions.

2. Meesmann dystrophy:

An autosomal dominant with the gene locus on 12q13 or 17q12.

▪ **Signs:**

Myriads of tiny intraepithelial eysts of uniform size but rariable density which are maximal centrally and extend towards but do not reach the limbus.

▪ **Histology:**

Irregular thickening of the corneal epithelium with numerous vaculated suprabasal cells.

▪ **Treatment:**

Not required.

• **Bowman layer dystrophies:**

1. Bowman layer type 1 (Reis – Bucklers dystrophy):

An autosomal dominant with the gene locus on 5q31.

▪ **Signs:**

1. Grey-white, fine, round and polygonal opacities in Bowman ayer, most dense centrally.
2. The changes increase indensity with age, resulting in a reticular pattern due to the laying down of irregular bands of collagen replacing Bowman layer.
3. Reduced corneal sensation.
4. Visual impairement may occur due to scarring at Bowman layer.

▪ **Histology:**

The new collagen stains blue with masson tricchrome, and Bowman layer is indistinct or absent.

▪ **Treatment:**

1. Excimer laser keratectomy.

2. Lamellar keratoplasty may be required.

3. Thiel-Behnke dystrophy (Bowman layer type 2):

An autosomal dominant with the gene locus on 10q24.

▪ **Signs:**

Similar to Reis-Buckler dystrophy except that the opacities assume more of a honeycomb pattern.

▪ **Histology:**

Similar to Reis-Buckler.

▪ **Treatment:**

May not be necessary because visual impairement is less than in Reis-Buckler.

4. Central schnyder (crystalline) dystrophy:

An autosomal dominant with the gene locus on 1p36 –p34.

▪ **Signs:**

1. Central, oval area of scintillating.

2. Subepithelial “crystalline” opacity in a generally hazy cornea.

▪ **Histology:**

Deposits of phospholipid and cholesterol.

▪ **Treatment:**

Excimer laser keratectomy.

• **Stromal dystrophies:**

1. Lattice dystrophy type 1 (Biber – Haab – Dimmer):

An autosomal dominant.

▪ **Signs:**

1. Anterior stromal dots.

2. Progression and coalescence into fine, spidery, branching lattice lines.

3. Deep and outward spread sparing the periphery.
4. Generalized haze progressively impairs vision and many obscure the lattice lines.

▪ **Histology:**

Amyloid which stains with Congo Red, exhibits metachromasia with crystal violet and birefringence under crossed polaroids.

▪ **Treatment:**

Penetrating or deep lamellar keratoplasty.

2. Lattice dystrophy type 2 (Meretoja syndrome):

An autosomal dominant.

▪ **Signs:**

Randomly scattered, short, fine lattice lines which are sparse, more delicate and more radially oriented than in type 1.

▪ **Histology:**

Amyloid deposits in the corneal stroma and other involved sites.

▪ **Treatment:**

Penetrating or deep lamellar keratoplasty.

3. Lattice dystrophy type 3 and 3A:

An autosomal recessive for type 3 and for 3A is autosomal dominant.

▪ **Signs:**

1. Thick, ropy, lines extending from limbus to limbus with minimal intervening haze.
2. There may be gross asymmetry or the lesions may be unilateral for atime.
3. Progression is rapid if the cornea is subjected to trauma.

▪ **Treatment:**

Penetrating or deep lamellar keratoplasty.

4. Granular dystrophy:

An autosomal dominant.

▪ **Signs:**

1. Small, white, sharply demarcated deposits resembling crumbs or snowflakes in the central anterior stroma.
2. Increase in number of lesions with deeper and autward spread but not reaching the limbus.
3. Gradual confluence causing impairment of visual oculty.

▪ **Histology:**

Amorphous hyaline deposits which stain bright red masson trichrome.

▪ **Treatment:**

1. Penetrating or deep lamellar keratoplasty.
2. Superficial recurlences may require excimer laser keratectomy.

5. Avellino dystrophy:

An autosomal dominant.

▪ **Signs:**

1. Superficial, fine, opacities that resemble rings, discs, stars or snowflakes, most dense centrally.
2. Deeper linear opacities reminiscent of lattice dystrophy.

▪ **Histology:**

Hyaline and amyloid in the stroma.

▪ **Treatment:**

Usually not required.

6. Macular dystrophy:

Is the least common stromal dystrophy in which a systemic inborn error of keratan sulphate metabolism has only corneal manifestations. It is an autosomal dominant.

▪ **Classification according to the presence or absence of antigenic keratan sulphate in the serum and cornea:**

1. Type I.
2. Type IA.
3. Type II.

▪ **Signs:**

1. Grayish-white, dense, focal, poorly delineated spots in the superficial cornea with mild diffuse stromal clouding.
2. Increasing opacification.
3. Eventual involvement of full-thickness stroma up to the limbus, associated with corneal thening.

▪ **Histology:**

Abnormally close packing of collagen in the corneal lamellae and abnormal aggregations of glycosominoglycans which stain with Alcian blue.

▪ **Treatment:**

Penetrating keratoplasty.

7. Gelatinous drop-like dystrophy:

This rare disorder is also known as familial subepithelial amyloidosis of the cornea.

This disorder is an autosomal ressisive.

▪ **Signs:**

1. Grey subepithelial nodules.
2. Gradual confluence, stromal involvement and increase in size to a nubbly, mulberry-like appearance.

▪ **Histology:**

Subepithelial and anterior stromal accumulation of amyloid.

▪ **Treatment:**

Superficial keratectomy.

• **Endothelial Keratitis:**

1. Fuchs endothelial dystrophies:

It is more common in women than men and is associated with a slightly increased prevalence of primary open, angle glaucoma.

It may occasionally be autosomal dominant although the majority are sporadic.

- **Signs:**

a. Stage 1:

Is characterized by a gradual increase of central guttate with peripheral spread and confluence, giving rise to a “beaten-metal” appearance.

b. Stage 2:

Is characterized by endothelial decompensation resulting in central stromal edema and blurred vision.

Initially worse in the morning, which clears later.

Epithelial edema develops when stromal thickness has increased by about 30%.

c. Stage 3:

Is characterized by persistent epithelial edema and results in bullous keratopathy which cause pain and discomfort on rupture, due to exposure of naked nerve endings.

Replacement of Bowman layer by degenerative pannus and gradual stromal opacification supervene.

- **Treatment:**

a. Hypertonicity of the tear film by sodium chloride 5% drops or ointment.

b. Bandage contact lenses.

c. Penetrating keratoplasty.

d. Conjunctival flaps and amniotic membrane transplants.

2. Posterior polymorphous dystrophy:

Is a rare, innocuous and symptomatic dystrophy in which corneal endothelial cells display characteristics similar to epithelium.

It is usually autosomal dominant.

- **Signs:**

Are subtle, consist of vesicular, band-like or geographic endothelial patterns which may be asymmetrical.

- **Treatment:**

Is not required.

3. Congenital hereditary endothelial dystrophy (CHED):

Is a rare dystrophy in which there is focal or generalized absence of corneal epithelium.

▪ **Classification:**

CHED 1 → an autosomal dominant.

CHED 2 → an autosomal recessive.

▪ **Signs:**

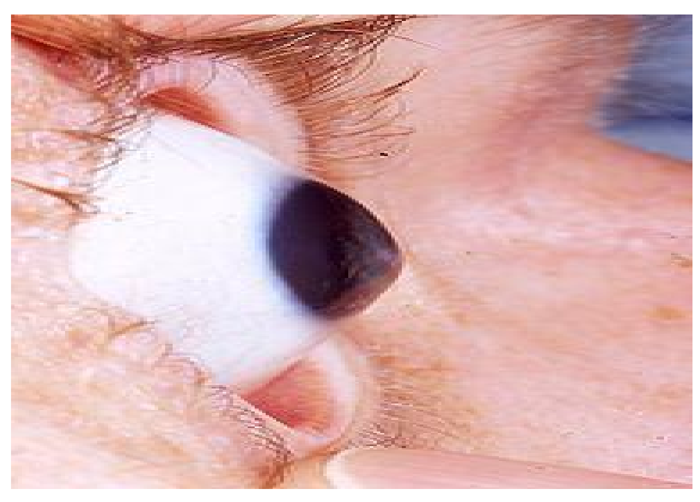
1. Bilateral, symmetrical, diffuse corneal edema.
2. Corneal appearance varies from a blue-grey, ground-glass appearance to total opacification.
3. Visual impairement is variable.

▪ **Treatment:**

Penetrating keratoplasty.

Corneal ectasias:

Keratoconus:



Is a progressive disorder in which the cornea assumes an irregular conical shape.

The role of heredity has not been clearly defined and most patients don't have a positive family history.

▪ **Association:**

1. Systemic disorders:

Include down, Turner, Ehlers-Danlos and Marfen syndromes, atopy, osteogenesis imperfecta and mitral valve prolapsed.

2. Ocular associations:

Include vernal keratoconjunctivities, blue sclera, aniridia, ectopra lentis, Leber congenital amaurosis and retinisis pegmentosa.rigid contact lens wear and eye rubbing have also been proposed as predisposing factors.

▪ **Classification according to morphology:**

1. Nipple cones:

Characterized by small size (5 mm) and steep curvature.

The apex is central or paracentral and displaced inferonasally.

2. Oval cones:

Are larger (5-6 mm), ellipsoid and commonly decentred inferotemporally.

3. Globus cones:

Are the largest (> 6 mm) may involve over 75% of the cornea.

▪ **Presentation:**

Is with unilateral impairment of vision due to progressive myopia and astigmatism, which subsequently becomes irregular.

The patient may complain of frequent changes in spectacle prescription or decreased tolerance to contact lens wear.

▪ **Signs:**

The hellmark is central or paracentral stromal thinning, apical protrusion and irregular astigmatism.

It can be graded by keratometry according to severity to:

a. Mild (< 48 D).

b. Moderate (48 – 54 D).

c. Sever (> 54 D).

- Early signs:

1. Oil droplet reflex in direct opthalmoscopy.
2. Irregular scissor reflex in retinoscopy.
3. Slit lamp shows very fine, deep stromal striae (vogt lines) which disappears with external pressure on the globe.
4. Prominent corneal nerves may also be present.
5. Irregular astigmatism.

- Later signs:

1. Progressive corneal thinning, to as little as one-third normal thickness, associated with poor visual acuity.
2. Munson sign (Bulging of the lower lid in down gaze).
3. Epithelial iron deposits (fleisctier ring).
4. Stromal scarring in sever cases.

- Acute hydrops:

Is an acute influx of aqueous into the cornea as a result of a rupture in Descement membrane.

1. Sudden drop in VA.
2. Discomfort.
3. Watering.

Although the break usually heals within 6-10 weeks and the corneal edema clears, a variable amount of stromal scarring may develop.

Initially it treated with hypertonic saline and patching or a soft bandage contact lens.

▪ **Management:**

1. Spectacle.
2. Rigid contact lenses.
3. Epikeratoplasty.
4. Keratoplasty.

Keratoglobus:

Is an extremely rare condition in which the entire cornea is abnormally thin.

- **Onset:**

At birth.

- **Signs:**

1. The cornea develops globular ectasia.
2. Corneal thinning.
3. Acute hydrops occurs, but less commonly than keratoconus.

- **Management:**

Sclera contact lenses.

Recurrent corneal erosion syndrome

- **Definition:**

Is a distressing condition characterized by disturbance of the epithelial basement membrane resulting in defective adhesion and recurrent break down of the epithelium.

- **Presentation:**

Is typically on waking with sudden pain, lacrimation, photophopia and blurred vision, which usually resolve spontaneously within a few hours.

- **Signs:**

a. Microerosions:

Are spontaneous small epithelial defects, associated with epithelial basement membrane dystrophy, which cause relatively mild symptoms.

b. Macroerosions:

Are extremely painful large epithelial defects with surrounding loosely adherent epithelium, most commonly associated with previous trauma.

Abrasions caused by a fingernail are particularly likely to progress to recurrent erosions.

▪ **Treatment of acute erosions:**

1. Topical treatment is with lubricants while the epithelium is healing.
2. Debridement is indicated in severe cases.

▪ **Prophylactic treatment:**

1. Topical lubricants
2. Extended-wear bandage contact lenses
3. Epithelial keratectomy
4. Anterior stromal micropuncture
5. Systemic tetracycline

Congenital corneal anomalies:

Megalocornea:

Is a rare, bilateral, non-progressive enlargement of the cornea affecting males.

Inheritance is x-linked recessive.

▪ **Signs:**

1. Corneal diameter is 13 mm or more
2. Very deep anterior champer
3. High myopia and astigmatism
4. Lens subluxation may occur due to zonular stretching

▪ **Systemic associations:**

Include Marfen, Apert, Ehlers-Danlos and Down syndromes, Osteogenesis imperfect, progressive facial hemiatrophy, renal carcinoma and mental handicap.

Microcornea:

Is a rare unilateral or bilateral condition.

Inheritance is usually AD.

▪ **Signs:**

1. The adult horizontal corneal diameter is 10 mm or less.

2. The anterior champer is shallow

▪ **Ocular associations:**

Include glaucoma (initially closed and later open angle), congenital cataract, cornea plana, leukoma, iris abnormalities, microphakia, optic nerve hypoplasia and hypermetropia.

▪ **Systemic associations:**

Include fetal alcohol, Turner, Ehlers-Dnlas, Weill-Marchesani, Waardenburg, Nance-Horan and Cornelia de lang syndromes.

Corneal ulcers:



A corneal ulcer forms when the surface of the cornea is damaged or compromised.

Causes:

May be sterile (no infecting organisms) or infectious.

Risk factors:

1. Contact lens (especially soft)

2. Bacterial ulcers may be associated with diseases that compromise the corneal surface, creating a window of opportunity for organisms to infect the cornea.

3. Patients with severely dry eyes, difficulty blinking, or are unable to care for themselves, are also at risk.

4. herpes simplex viral infections, inflammatory diseases, corneal abrasions or injuries, and other systemic diseases.

Signs and Symptoms:

The symptoms associated with corneal ulcers depend on whether they are infectious or sterile, as well as the aggressiveness of the infecting organism.

• Red eye

• Severe pain (not in all cases)

• Tearing

• Discharge

• White spot on the cornea, that depending on the severity of the ulcer, may not be visible with the naked eye

• Light sensitivity

Diagnosis:

- slit lamp microscope.

- Special types of eye drops containing dye such as fluorescein

- If an infectious organism is suspected, the doctor may order a culture.

Treatment:

The course of treatment depends on whether the ulcer is sterile or infectious. Bacterial ulcers require aggressive treatment. In some cases, antibacterial eye drops are used every 15 minutes. Steroid medications are avoided in cases of infectious ulcers. Some patients with severe ulcers may require hospitalization for IV antibiotics and around-the-clock therapy. Sterile ulcers are typically treated by reducing the eye's inflammatory response with steroid drops, anti-inflammatory drops, and antibiotics.

Chapter Five
Lacrimal gland, sac & duct disease

Lacrimal gland, sac and duct disease

Applied anatomy:

1. The punctal:

Are located at the posterior edge of the lid margin.

2. The canaliculi:

Pass vertically from the lid margin for about 2 mm. they then turn medially and run horizontally for about 8 mm to reach the Lacrimal sac.

The superior and inferior canaliculi most often unit to form the common canaliculus.

3. The Lacrimal sac:

Is about 10 mm long and lies in the Lacrimal fossa between the anterior and posterior Lacrimal crests.

4. The nasolacrimal duct:

Is about 12 mm long and is the inferior continuation of the Lacrimal sac.

Obstruction of tear drainage:

Nasolacrimal duct obstruction

Acquired nasolacrimal duct obstruction:

- **Causes:**

1. Idiopathic stenosis
2. Naso-orbital trauma
3. Wegener granulomatosis
4. Infiltration by nasopharyngeal tumours

- **Treatmenet:**

1. Complete obstruction is treated by DCR
2. Incomplete obstruction may response to intubation of the Lacrimal system with silicon tubes.

Congenital nasolacrimal duct obstruction:

- **Signs:**

1. Epiphora and matting of lashes
2. Gentle pressure over the Lacrimal sac causes reflux of purulent material from the punctal.
3. Acute dacryocystitis is uncommon

- **Treatment:**

1. Massage of the Lacrimal sac
2. Probing of the Lacrimal system should be delayed until the age of 12 months

Congenital dacryocele:

Is a collection of amniotic fluid or mucus in the Lacrimal sac caused by an imperforate Hasner valve.

- **Symptoms:**

Is a prenatal with a bluish cystic swelling at or below the medial canthal area, accompanied by epiphora.

- **Signs:**

A tense Lacrimal sac which is initially filled with mucus but may become secondarily infected.

- **Treatment:**

It is initially conservative but if fails probing should not be delayed.

Infections of Lacrimal passage:

Chronic canaliculitis:

Is an uncommon condition, frequently caused by Actinomyces.

- **Symptoms:**

Is with unilateral epiphora associated with chronic mucopurulent conjunctivitis

• **Signs:**

1. Pericanalicular inflammation is characterized by edema of the canaliculus and a "pouting" punctum.
2. Concretions consisting of sulphur granules are expressed on canalicular compression with a glass rod

• **Treatment:**

1. Topical antibiotics
2. Canaliculotomy

Dacryocystitis:

Infection of the Lacrimal sac, is usually secondary to obstruction of the nasolacrimal duct.

It is most commonly caused by staphylococci.

• **Classifications:**

1. Acute dacryocystitis:

- Symptoms:

Is subacute onset of pain, redness and swelling at the medial canthus and epiphora.

- Signs:

A very tender, red, tense swelling at the medial canthus which may be associated with preseptal cellulitis in sever cases.

- Treatment:

1. Local warmth and oral antibiotics.
2. Incision and drainage
3. DCR

2. Chronic dacryocystitis:

- Symptoms:

Epiphora which may be associated with a chronic or recurrent unilateral conjunctivitis.

- Signs:

1. A painless swelling at the inner canthus caused by a mucocele.
2. Obvious swelling may be present
3. Pressure over the sac commonly results in reflux of mucupurulent material through the canaliculi.

- Treatment:

DCR

Dry eye

• A deficiency of Lacrimal secretion occurs with age and result in dry eye.

Symptoms:

1. Grittiness
2. Burning
3. Photophobia
4. Heaviness of the lids
5. Ocular fatigue
6. In severe cases visual acuity may be reduced by corneal damage

Signs:

1. Staining of the eyelid with fluorescien will show small dots of fluorescien over the exposed corneal and conjunctival surfaces
2. In severe cases tags of abnormal mucus may attach to the corneal surface causing pain due to tugging on these filaments during blinking.

Treatment:

1. Artificial tears
2. Shielded spectacles
3. In severe cases: may be necessary to occlude the Punta with plugs or with surgery.

Epiphora

Is overflow of tears onto the face. A clinical sign or condition that constitutes insufficient tear film drainage from the eyes in that tears will drain down the face rather than through the nasolacrimal system.

Etiology:

1. trichiasis
2. entropion
3. Ectropion
4. punctal, canalicular or nasolacrimal duct obstruction.
5. aging (a spontaneous process)
6. infection (ie. dacryocystitis)
7. rhinitis
8. failure of the nasolacrimal duct to open.

Diagnosis:

1. history presentation
2. observation of the lids
3. Fluorescein dye can be used to examine for punctal reflux by pressing on the canaliculi in which the clinician should note resistance of reflux as it irrigates through the punctum into the nose

Management:

1. lid repair is indicated
2. Punctal irrigation is also required
3. nasolacrimal duct probe is used and a tube replacement

Inadequate Nucus Production

destruction of the goblet cells occurs in most forms of dry eye, but particularly in cicatricial conjunctival disorders.

There may be both an aqueous and mucin deficiency and problems due to lid deformity and Trichiasis.

Symptoms:

1. Grittiness

2. Burning

3. Photophobia

4. Heaviness of the lids

5. Ocular fatigue

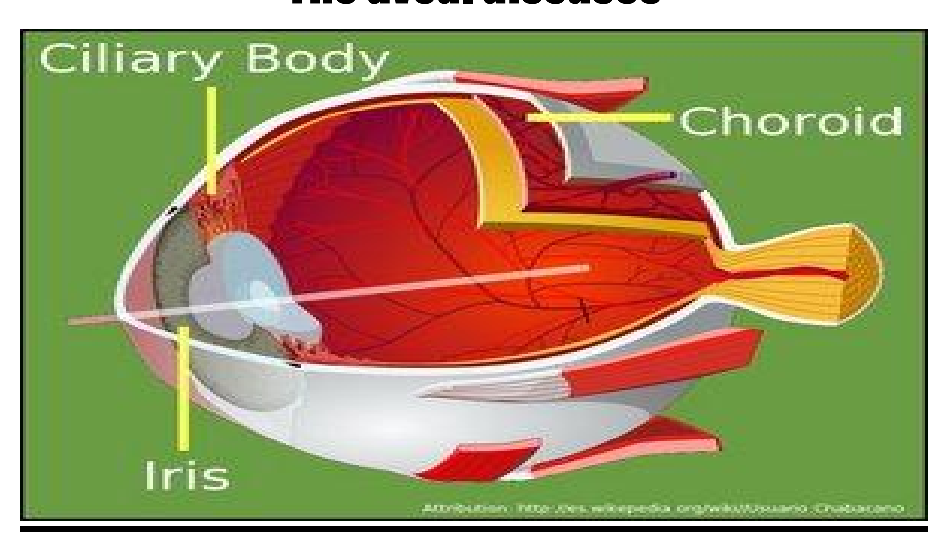
6. In severe cases visual acuity may be reduced by corneal damage

Treatment:

Artificial lubricants.

Chapter Six
The Uveal Diseases

The uveal diseases



Uveitis

Inflammation of the uveal tract (iris, ciliary body, choroid)

Anatomical classification:

1. Inflammation of the iris:
    - Accompanied by increased vascular permeability.
    - Termed iritis or anterior uveitis.
    - White cells circulating can be seen in the aqueous humour with slit lamp.
2. Inflammation of the pars plana (posterior ciliary body):
    - Termed cyclitis or intermediate uveitis.
3. Inflammation of the posterior segment (posterior uveitis):
    - Results in inflammatory cells in the vitreous gel.
    - May also be an associated choroidal or retinal inflammation.

Symptoms:

1. Ocular pain
2. Photophobia
3. Blurring of vision

4. Redness of the eye.

• Posterior uveitis may not be painful.

Associated systemic diseases:

1. Respiratory symptoms.
2. Skin problems
3. Syphilis

Signs:

1. Visual acuity may be reduced.
2. The eye will be inflamed mostly around the limbus.
3. Inflammatory cells may be visible on the endothelium of the cornea.
4. Slit lamp examination will reveal aqueous cells and flare
5. The vessels on the iris may be dilated
6. The iris may adhere to the lens
7. The IOP may be elevated
8. There may be cells in the vitreous
9. There may be retinal or coroidal inflammation
10. Macular edema may be present.

Treatment is aimed at:

1. Relieving the pain and inflammation
2. Preventing damage to ocular structures.

Treatment:

1. Steroids (eye drops, injection, systemic)
2. In anterior uveitis, dilating the pupil relieves the pain from ciliary spasm.
3. Antiviral or antibiotics medication may be required.

Some rare and sever forms of uveitis may require treatment with systemic immunosuppressive drugs

Chapter Seven
Glaucoma

Glaucoma



Definition:

Are mixed group of disorders that have some common features:

1. Optic disc cupping
2. Visual field loss
3. Raised IOP

- Raised IOP without optic disc damage and visual field loss is called ocular hypertension.
- Glaucoma in the absence of high pressure is known as low or normal tension glaucoma.

Classifications:

- Primary versus secondary
- Open versus closed

• Congenital versus acquired

Primary open angle glaucoma

Pathogenesis:

1. Impaired drainage of aqueous humour which causes raise of IOP, which transmitted to the optic disc where nerve fiber damage occur.
2. Impairment of the optic nerve blood supply
3. Aspects of optic nerve head structure.

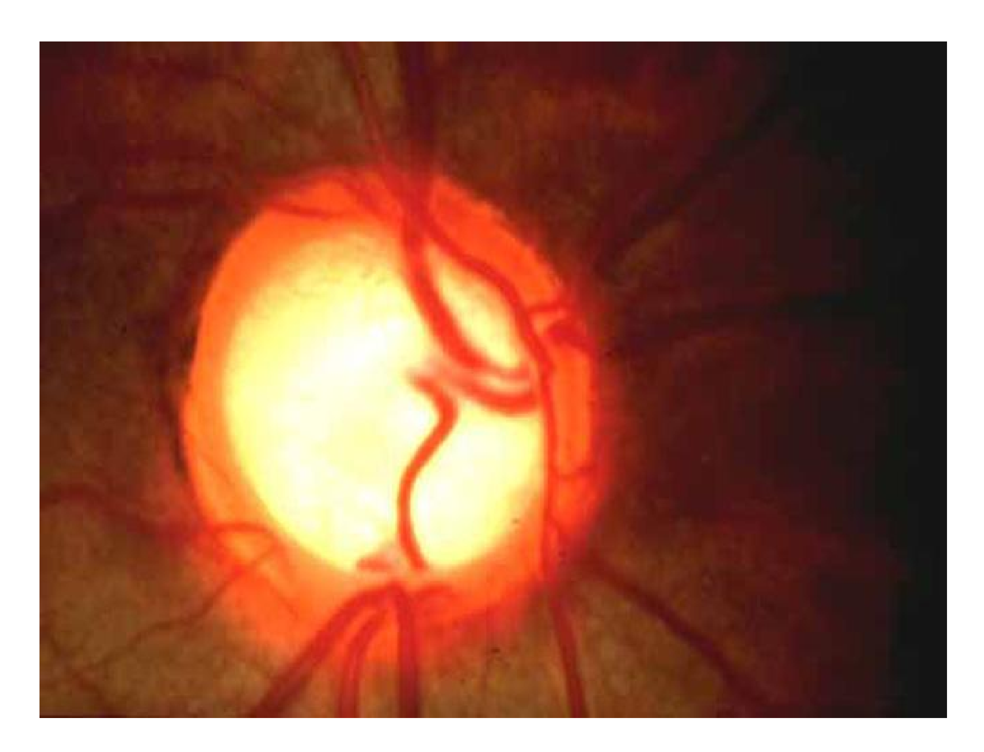
- The 2nd and 3rd points are called glaucoma when the IOP is normal or low.

Risk factors:

1. Genetic
2. Increasing age
3. Diabetes mellitus
4. Myopia
5. Black race

Clinical features:

1. No symptoms until it is so advanced
2. The central vision is threatened
3. Does not present with head or eye pain
4. Does not present with loss of visual acuity.
5. Raised IOP
6. Optic disc cupping



1. Peripheral visual field loss.

Examination:

1. Tonometer (for measuring IOP): the normal IOP 11-21 mmHg, the average is 15.5 mmHg.
2. Optic disc examination is essential:

The normal optic disc contains:

- Nerve fibers (the neuroretinal rim, which is pink)
- An area without nerve fibers (the optic disc cup, which is white)
- Blood vessels.

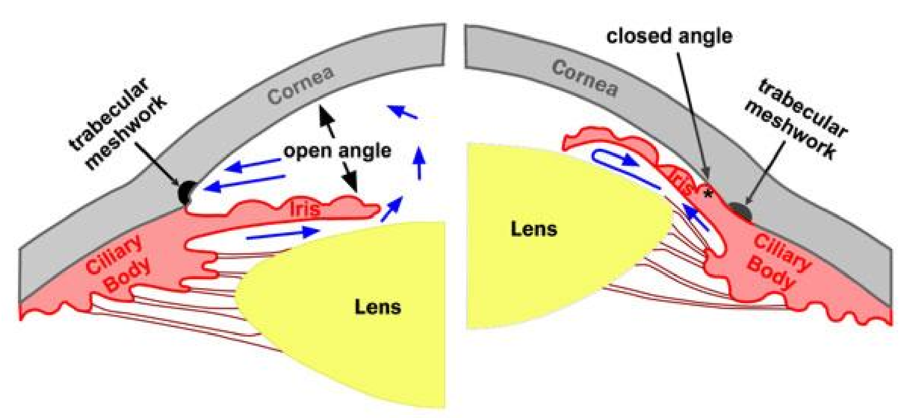
As nerve fibers are damaged and lost, the proportion of pink neuroretinal rim diminishes, the rim becomes pale and the cup enlarges.

3. Examine the iridocorneal angle with the gonioscopy lens to confirm that an open angle is present.

Management:

1. Lowering of the IOP by suppression of the aqueous humour formation (drops and tablets)
2. Increase in aqueous outflow (drops, drainage surgery)

Angle closure glaucoma



- Typically primary.

Clinical features:

1. Pain, nausea, vomiting
2. Loss of vision
3. Haloes
4. Red eye (usually unilateral)
5. Cloudy cornea (caused by corneal edema)
6. Oval, nonreactive pupil
7. Los of red reflex.

Management:

1. Treat the elevated IOP urgently, with topical and systemic aqueous suppressants (beta-blockers and acetazolamide)
2. The pupil block can be reversed by pilocarpine.
3. Systemic administration of analgesics and anti-emetics is welcome to the patient.

4. Once the acute attack has resolved, treatment to prevent recurrence and to prevent involvement of the at-risk fellow eye must be undertaken. this requires a laser iridotomy, which allows aqueous humour to pass from the posterior to the anterior chamber, by passing the pupil.

Secondary glaucoma

May be open or closed

May present acutely with pain and visual loss, or insidiously like primary open glaucoma

Treatment is aimed first the cause if possible, followed by standard glaucoma treatment.

Causes:

• Inflammation

• Pseudoexfoliation

• Pigment dispersion

• Aphakia

• Lens abnormalities

• Iris neovascularisation (rubeosis)

• Steroid therapy (usually topical)

• Uveitis if chronic.

Management:

• The primary cause should be treated

• Surgical treatment.

Congenital glaucoma

- Present at birth, in infancy or even in childhood.
- Primary congenital glaucoma caused by abnormal development of the anterior chamber angle.

- Secondary congenital glaucomas are uncommon.

Clinical features:

- The infant eyeball unlike adults can enlarge with elevation of IOP (buphthalmos) (ox eye)
- The cornea becomes hazy
- The eye waters

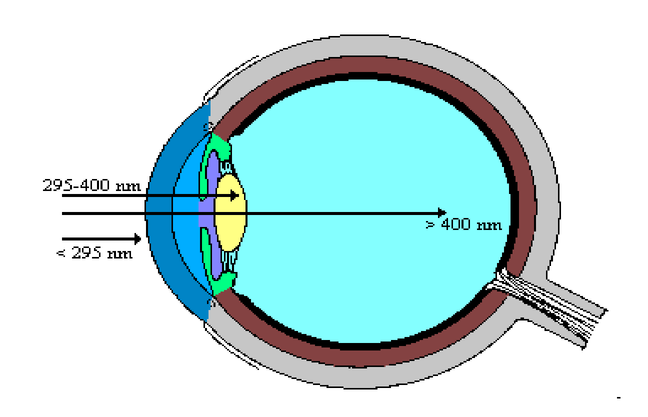
Treatment:

Surgery but often fails.



Chapter Eight
Lens diseases

Lens diseases



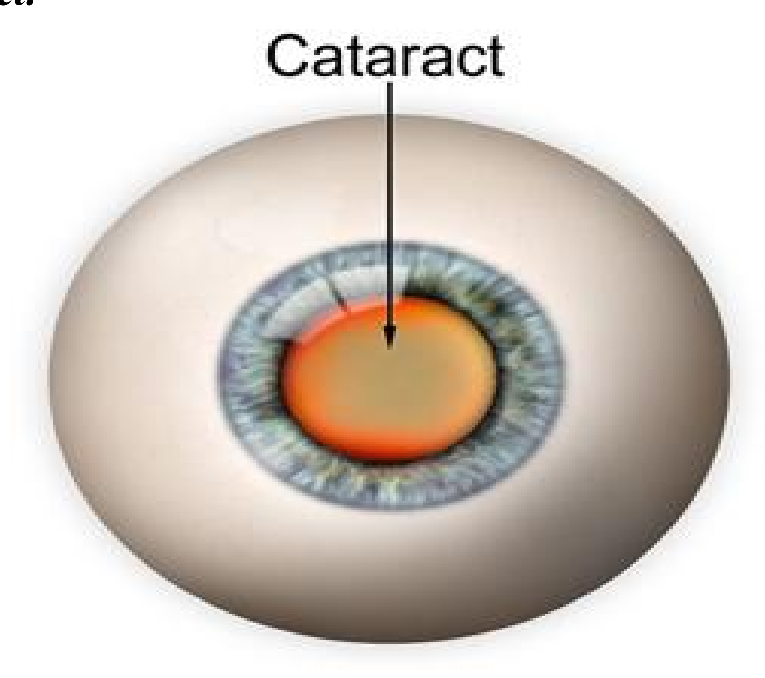
Applied anatomy:

The crystalline lens is a biconvex, avascular, transparent structure enclosed by a capsule.

The capsule responsible for moulding the lens substance during accommodation.

A ring of zonular fibers suspends the lens from the ciliary body.

Cataract:



Classification:

a. Acquired

b. Congenital

Acquired cataract:

Age related cataract:

• **Morphological classification:**

1. Subcapsular cataract:

a. Anterior Subcapsular cataract:

Lies directly under the lens capsule and is associated with fibrous metaplasia of the lens epithelium.

b. Posterior Subcapsular cataract:

Lies just infront of the posterior capsule.

A posterior Subcapsular opacity has a more profound effect on vision than a comparable nuclear or cortical cataract.

Patients are particularly troubled under conditions of miosis, such as produced by headlights of oncoming cars and bright sunlight.

Near vision is also frequently impaired more than distance vision.

2. Nuclear cataract:

Involving the lens nucleus.

It is often associated with myopia due to the increase in the refractive index, and also associated with increased spherical aberration.

Nuclear sclerosis is characterized in its early stages by a yellowish hue due to the deposition of urochrome pigment, when advanced the nucleus appear brown.

3. Cortical cataract:

May involve the anterior, posterior or equatorial cortex. The opacities starts as clefts and vacuoles between lens fibers due to hydration of the cortex.

Subsequent opacification results in typical wedge-shaped or radial spoke-like opacities.

4. Christmas tree cataract:

Which is uncommon.

Is characterized striking, polychromatic, needle-like deposits in the deep cortex and nucleus which may be solitary or associated with other opacities.

- **Classification according to maturity:**

1. An immature
2. A mature
3. A hyper mature
4. A morgagnian

Presenile cataract:

Cataract may develop early in the following condition:

1. Diabetes mellitus:

a. Classical diabetic cataract

b. Age related cataract (occurs earlier)

c. Premature presbyopia

2. Myotonic dystrophy:

About 90% of patients develop visually innocuous, fine cortical iridescent opacities in the third decade, which involve into visually disabling stellate posterior Subcapsular cataract by the fifth decade.

Occasionally cataract may antedate myotonia.

3. Atopic dermatitis:

About 10% of patients with sever atopic dermatitis develop cataract in the second to fourth decade.

a. sheild-like dense anterior Subcapsular cataract.

b. Posterior Subcapsular cataract.

4. Neurofibromatosis type 2:

Is associated with posterior Subcapsular or posterior cortical opacities.

Traumatic cataract:

Trauma is the most common cause of unilateral cataract in young individuals.

- **Causes:**

1. Direct penetrating injury to the lens
2. Concussion
3. Electric shock and lightning are rare
4. Lionizing radiation to ocular tumor
5. IR radiation

Drug-induced cataract:

1. Steroids:

Both systemic and topical.

The lens opacities are initially posterior Subcapsular, later the anterior Subcapsular region become affected.

2. Chlorpromazine:

May cause the deposition of innocuous, fine, stellate, yellowish-brown granules on the anterior lens capsule within the papillary area.

Diffuse, granular deposits on the corneal endothelium and in the deep stroma may also occur.

3. Busulphan (myleran):

Used in the treatment of chronic myeloid leukemia.

4. Amiodarone:

Used in the treatment of cardiac arrhythmias.

Causes anterior Subcapsular cataract.

5. Gold:

Used in the treatment of rheumatoid arthritis.

Causes anterior Subcapsular cataract.

6. Allopurinol:

Used in the treatment of hyperuricaemia and chronic goul.

Secondary cataract:

Develops as a result of some other primary ocular disease:

1. Chronic anterior uveitis
2. Acute congestive angular-closure glaucoma
3. High (pathological) myopia
4. Hereditary fundus dystrophies

Treatment of cataract:

Cataract surgery:

1. Extracapsular cataract extraction (ECCE)
2. Phacoemulsification (phaco)

Congenital cataract:

Occur in about 3:10000 live births, two third of cases are bilateral.

The most common cause is genetic mutation, usually AD. Other causes include chromosomal abnormalities such as Down syndrome, metabolic disorders and rebulla infection.

Congenital cataract may also occur as part of a complex developmental disorder of the eye such as aniridia.

Cataract without systemic associations:

• **Isolated hereditary cataract:**

The mode of inheritance is most frequently AD but may be AR or x-linked.

- ***Morphological classification:***

1. Zonular cataract:

In which the opacity occupies a discrete zone in the lens may be:

a. Nuclear

b. Lamellar

c. Capsular

d. Sutural

2. Polar cataract:

In which the opacities occupy the Subcapsular cortex at the anterior or posterior pole of the lens.

a. Anterior polar cataract

b. Posterior polar cataract

• **Systemic associations:**

- Metabolic:

1. Glactosaemia

2. Glactokinase deficiency

3. Lowe (oculocerebrorenal) syndrome

4. Hypoparathyroidism

5. Pseudohypoparathyroidism

6. Mannosidosis

- Prenatal infections:

1. Congenital rebulla

2. Toxoplasmosis

3. Cytomegalovirus

4. Herpes simplex

5. Varicella

- Chromosomal abnormalities:

1. Down syndrome

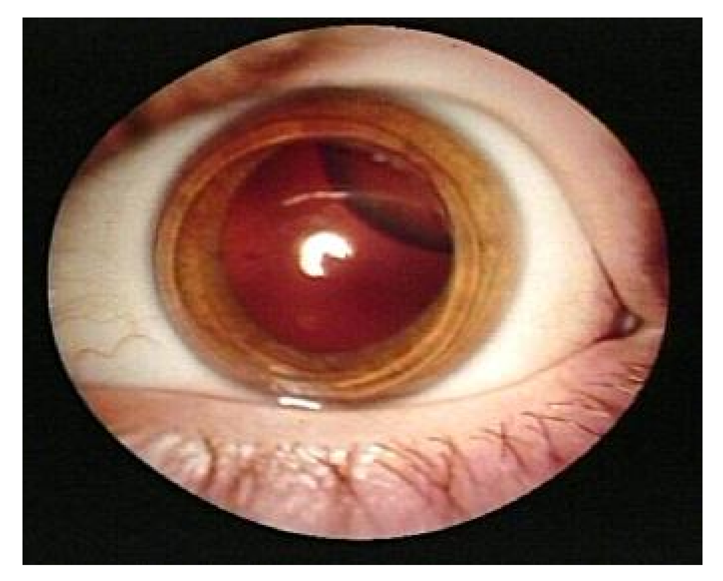
2. Hallerman-Streiff-Francois syndrome

3. Nance-Horan syndrome

• **Management of congenital cataract:**

Surgery

Ectopia lentis



Weakness of the zonule causes lens displacement. The lens takes up a more rounded form and the eye becomes more myopic.

This may be seen in:

1. trauma
2. inborn errors of metabolism
3. certain syndromes

treatment:

1. the irregular myopia can be corrected optically
2. sometimes an Aphakia correction may be required if the lens is substantially displaced from the visual axis.
3. Surgical removal may be indicated if the displaced lens has caused a secondary glaucoma

Aphakia

The absence of the lens of the eye.

Causes:

1. Surgical removal
2. Perforating wound or ulcer
3. Congenital

It causes:

1. Loss of accommodation
2. Hyperopia

Complications:

1. Detachment of the vitreous
2. Detachment of the retina
3. Glaucoma

Note: aphakic patients are reported to be able to see UV wavelength that are normally excluded by the lens.

Treatment:

1. Glasses
2. Contact lenses
3. Artificial lens (Pseudophakia)

Chapter Nine
Optic nerve disorders

Optic nerve disorders

Applied anatomy:

1. Afferent fibers: the optic nerve carries about 1.2 million afferent nerve fibers.
2. Oligodendrocytes: provides axonal myelination
3. Microglia: line the space between axons and other structures.
4. Surrounding sheaths:
   a. Pia mater
   b. Subarachnoid space
   c. Other sheath comprises the arachnoid mater and the tougher dura mater

Anatomical subdivision:

1. Intraocular segment:

(optic disc, nerve head) is the shortest, being 1 mm deep and 1.5 mm in vertical diameter.

In relation to the lamina cribrosa the intraocular segment can be further subdivided into 3 zones:

- The prelaminar zone
- The laminar zone
- The post laminar zone

2. Intraorbital segment:

Is 25-30 mm long and extends from the globe to the optic foramen at the orbital apex. Its diameter is 3-4 mm because of the addition of the myelin sheaths to the nerve fibers.

At the orbital apex the nerve is surrounded by the tough fibrous annulus of zinn.

3. Intracanalicular segment:

Traverses the optic canal and measures about 6 mm.

4. Intracranial segment:

Joins the chiasm and varies in length from 5 to 16 mm (avg 10 mm)

Optic atrophy:

An important sign of advanced optic nerve disease, may be primary or secondary.

1. Primary optic atrophy:

This occur without antecedent swelling of the optic nerve head.

It may be caused by lesions affecting the visual pathways from the reterolaminar portion of the optic nerve to the lateral geniculate body.

Lesions anterior to the optic chiasm result in unilateral optic atrophy, whereas those involving the chiasm and optic tract will cause bilateral optic atrophy.

• **Causes:**

1. Following reterobulbar neuritis
2. Compressive lesions such as tumours and aneurysms
3. Hereditary optic neuropathies
4. Toxic and nutritional optic neuropathies

• **Signs:**

1. Pale, flat disc with clearly delineated margins
2. Reduction in number of small blood vessels on the disc surface
3. Attenuation of parapapillary blood vessels and thinning at the retinal nerve fiber layer
4. The atrophy may be diffuse or sectoral depending on the cause and level of the lesion

2. Secondary optic atrophy:

Is preceded by swelling of the optic nerve head

• **Causes:**

1. Chronic papilloedema
2. Anterior ischemic optic neuropathy
3. Papillitis

• **Signs:**

1. White or dirty grey, slightly raised disc with poorly delineated margins due to gliosis
2. Reduction in number of small blood vessels on the disc surface.

Optic neuritis:

Is an inflammatory, infective or demylineating process affecting the optic nerve.

• Ophthalmoscopic classification:

1. Retrobulbar neuritis:

The optic disc appearance is normal, at least initially.

It is the most frequent type in adults, and is frequently associated with MS.

2. Papillitis:

The pathological process affects the optic nerve head primarily, or secondary to contiguous retinal inflammation.

Characterized by variable hyperaemia and edema of the optic disc, which may be associated with parapapillary flame-shaped haemorrhages.

Cells in the posterior vitreous may be seen.

It is the most common type in children, although it can also affect adults.

3. Neuroretinitis:

Characterized by Papillitis in association with inflammation of the retinal nerve fiber layer.

A macular star figure composed of hard exudates may not be present initially, but become apparent within a few days or weeks.

In some cases there is associated parapapillary retinal edema and serous elevation of the macula.

It is the least common type and is the most frequently associated with viral infections and cat-scratch fever.

Other causes include syphilis and lyme disease.

In most cases it is self-limiting disorder which resolve within 6-12 months.

• Aetiological classifications:

1. Demyelinating:

Which is the most common cause.

2. Parainfections:

Which may follow a viral infection or immunization.

3. Infectious:

Which may be sinus-related or associated with cat-scratch fever, syphilis, lyme disease, cryptococcal meningitis in patients with AIDs and herpes zoster.

4. Autoimmune:

Which may be associated with systemic autoimmune disease.

Alcohol-tobacco amblyopia:

Typically affects heavy drinkers and cigar and pipe smokers who are deficient in protein and the B vitamins.

Most patients have neglected their diet, obtaining their calories from alcohot instead.

- **Symptoms:**

Is insidious onset, progressive, bilateral, usually symmetrical visual impairment associated with dyschromatopsia.

- **Signs:**

1. The optic disc at presentation are normal in most cases
2. Some patients show subtle temporal pallor, splinter-shaped haemorrhages on or around the disc, or minimal disc edema.

- **Visual field defects:**

Are bilateral, relatively symmetrical, centrocaecal scotomas.

The margins of the defects are difficult to define with a white target but are easier to plot and larger when using a red target.

- **Treatment:**

1. Weekly injections of 1000 units of hydroxocobalamin for 10 weeks.
2. Multivitamins

Drug-induced optic neuropathies:

1. Ethambutol:

Is used in combination with isoniazid and rifampicin in the treatment of tuberculosis.

- **Symptoms:**

Symmetrical insidious visual impairment associated with dyschromatopsia.

- **Signs:**

Normal or slightly swollen optic discs with splinter-shapped haemorrhages.

- **Visual field defects:**

Consist of central or centrocaecal scotomas.

Although bitemporal or peripheral construction may occur.

2. Amiodarone:

Is used to treat cardia arrhythmias.

- **Symptoms:**

Insidious unilateral or bilateral visual impairment.

- **Signs:**

Bilateral optic disc swelling that stabilizes within several months of discontinuing medication

- **Visual field defects:**

May be mild and reversible or sever and permanent.

Papilloedema:

Is swelling of the optic nerve head, secondary to raised intracranial pressure.

It is nearly always bilateral, and may be asymmetrical.

All patients with Papilloedema should be suspected of having an intracranial mass unless provide otherwise.

• ***Clinical features:***

1. Early Papilloedema:

- Visual symptoms are absent and VA is normal
- Optic disc show hyperaemia and mild elevation
- The disc margins appear indistinct, and swelling of the parapapillary retinal nerve fiber layer develops.
- There is loss of previous spontaneous venous pulsation.

2. Established Papilloedema:

- Transient visual obstructions may occur in one or both eyes, lasting a few seconds, often on standing.
- Visual acuity is normal or reduced
- Optic disc show sever hyperaemia, and moderate elevation with indistinct margins, which may initially be asymmetrical.
- The optic cup and the small vessels on the disc are obstructed.
- Venous engorgement, parapapillary flame-shaped haemorrhage and frequently also cotton wool spots may be seen.
- As the swelling increases, the optic nerve head appears enlarged
- Hard exudates may radiate from the center of the fovea in the form of a "macular fan".

3. Long-standing (vintage) Papilloedema:

- VA is variable and the VF being to constrict.
- Optic discs are markedly elevated with a "champagne cork" appearance.
- Cotton wool spots and haemorrhages are absent.
- Opticociliary shunts and drusen-like crystalline deposits may be present on the disc surface.

4. Atrophic Papilloedema (secondary optic atrophy):

- VA is severely impaired
- The optic discs are dirty grey, slightly elevated, with few crossing blood vessels and indistinct margins.
- Congenital optic nerve anomalies with neurological associations

Optic disc drusen (hyaline bodies):
composed of hyaline-like calcific material within the substance of the optic nerve head.

• Clinical features:

1. Buried drusen:

In early childhood drusen may be difficult to detect because they lie deep beneath the surface of the disc.

In this setting the appearance may mimic Papilloedema.

Signs suggestive of disc drusen are:

- Elevated disc with a scalloped margin without a physiological cup
- Anomalous vascular patterns including early branching, increased number of major retinal vessels and vascular tortousity.

2. Exposed drusen:

During the early teens drusen usually emerge at the surface of the disc as waxy pearl-like irregularities.

• Complications:

Are uncommon.

- A small minority of patients develop visual impairment.
- Occasionally a progressive but limited loss of VF may occur.

• Associations:

Retinitis pigmentosa, angioid streaks and Allagille syndrome.

Optic disc coloboma:

Result from incomplete closure of the Choroidal fissure.

Mostly sporadic, although AD inheritance may occur.

• **Signs:**

- VA is often decreased
- The inferior neuroretinal rim is thin or absent
- The optic disc may be enlarged

• **Visual field:**

Shows a superior defect

• **Ocular associations:**

- Microphthalmos
- Colobomas of iris, ciliary body and fundus

• Complications:

- Serous retinal detachment at the macula
- Progressive enlargement of the excavation
- Rhegmatogenous retinal detachment may occur in eyes with associated choroidoretinal colobomas.

Morning glory anomaly:

Usually unilateral sporadic condition. Bilateral cases may be hereditary.

• **Signs:**

- VA is usually very poor
- The disc is enlarged and manifests a funnel shaped excavation
- A central core of whitish glial tissue
- The disc is surrounded by an elevated annulus of choroidoretinal pigmentary disturbance
- The blood vessels emerge from the rim of the excavation in a radial pattern like the spokes of a wheel.

• **Complication:**

Retinal detachment.

Optic nerve hypoplasia:

Is characterized by a diminished number of nerve fibers

• **Signs:**

- VA can vary from normal to NLP.
- The disc is small and grey and is surrounded by a yellow halo of hypopigmentation

- The distance from the fovea to the temporal border of the disc often equals or exceeds three times the disc diameter

Aicardi syndrome:

Is very rare x-linked dominant disorder.

Usually bilateral but often asymmetrical

• **Signs:**

- Multiple depigmented clustered around the disc are pathognomonic
- Congenital disc anomalies include coloboma, hypoplasia and pigmentation.

Chapter Ten
Scleral disorders

Scleral disorders

Applied anatomy:

The sclera stroma is composed of collagen bundles of varying size and shape.

The inner layer of the sclera (lamina fusca) blends with the suprachoroidal and supraciliary lamellae of uveal tract.

Anteriorly the episclera consists of a dense, vascular connective tissue which lies between the superficial sclera stroma and Tenon capsule.

The three vascular layers that cover the anterior sclera are as follows:

1. The conjunctival vessels
2. The vessels within the Tenon capsule
3. The deep vascular plexus

Episcliritis:

Is a common, benign, self-limiting and frequently recurrent disorder. Episcliritis may be simple or nodular.

• **Symptoms:**

Unilateral redness associated with mild discomfort, tenderness and watering.

• **Signs:**

a. Simple Episcliritis:

Characterized by sectoral, or diffuse redness.

It usually resolves spontaneously within 1-2 weeks.

b. Nodular Episcliritis:

Characterized by a localized, raised, congested nodule which takes longer to resolve.

- A thin slit-lamp section shows that the anterior sclera surface is not raised, indicating that the sclera is not swollen.

- Following recurrent attacks, the superficial sclera lamellae may become rearranged into more parallel rows, making the sclera appear more translucent.

• **Treatment:**

1. Simple lubricants or vasoconstrictors
2. Topical steroids
3. Oral non-steroidal anti-inflammatory drugs (NSAIDs)

Scleritis:

Is characterized by edema and cellular infiltration of the entire thickness of the sclera.

• **Associations:**

1. Rheumatoidal arthritis
2. Wegener granulomatosis
3. Relapsing polychondritis
4. Polyarteritis nodosa.

• **Causes:**

1. Ocular surgery
2. Corneal ulcer
3. Trauma
4. Excision of a pterygium

• Causative organisms:

1. Pseudomonas aeruginosa
2. Streptococcus pneumonia
3. Staphylococcus aureus
4. Varicella zoster virus

• **Classification:**

1. Anterior Scleritis:

a. Non-necrotizing

b. Necrotizing

2. Posterior Scleritis

• Anterior non-necrotizing Scleritis:

- **Signs:**

a. Diffuse Scleritis:

- Characterized by widespread inflammation involving a sector or the entire anterior sclera.

- Distortion of the normal radial vascular pattern.

b. Nodular Scleritis:

- May resemble nodular episcleritis. However, the scleral nodule cannot be moved over the underlying tissue.

- Visual impairment.

- **Treatment:**

1. Oral NSAIDs
2. Oral Prednisolone
3. Combined therapy with an NSAIDs and lower dose of steroid
4. Subconjunctival steroid injection.

• Anterior necrotizing Scleritis with inflammation:

- **Signs:**

1. Congestion of the deep vascular plexus
2. Vascular distortion and occlusion
3. Scleral necrosis which may be associated with overlying conjunctival ulceration
4. Gradual spread of necrosis which may coalesce with other separate necrotic areas
5. Upon resolution a bluish tinge appears due to increased visibility of underlying uvea

- **Symptoms:**

1. Gradual onset of pain
2. Localized redness

- **Complications:**

1. Staphyloma formation
2. Anterior uveitis

▪ **Treatment:**

1. Oral Prednisolone

2. Immunosuppressive agents

3. Combined therapy (pulsed intravenous methylprednisolone and cyclophosphamide)

• Anterior necrotizing Scleritis without inflammation (scleromalacia perforans):

▪ **Signs:**

1. A symptomatic yellow necrotic sclera patches in un-inflamed sclera.

2. Enlargement, spread and coalescence

3. Progressive exposure of underlying uvea as a result of scleral thinning.

4. Staphyloma formation

▪ **Treatment:**

Is ineffective.

• Posterior Scleritis:

▪ **Symptoms:**

1. Pain

2. Visual impairment

▪ **Signs:**

1. External signs include:

a. Lid edema

b. Proptosis and ophthalmoplegia

c. Anterior Scleritis

2. Fundus findings include:

a. Disc swelling

b. Macular edema

c. Choroidal folds

d. Exudative retinal detachment

e. Ring Choroidal detachment

f. Subretinal lipid exudation

- **Treatment:**

- In elderly patients with associated systemic diseases

a. Oral Prednisolone

b. Immunosuppressive agents

c. Combined therapy (pulsed intravenous methylprednisolone and cyclophosphamide)

- In young patients without associated systemic diseases:
NSAIDs

Chapter Eleven
Orbital disorders

Orbital disorders

Applied anatomy:

The orbit is a pear-shaped cavity, the stalk of which is the optic canal.

1. The roof:

   Consist of two bones: lesser wing of the sphenoid and the orbital plate of the frontal

2. The lateral wall:

   Consist of two bones: greater wing of the sphenoid and zygomatic

3. The floor:

   Consist of three bones: zygomatic, maxillary and palatine

4. The medial wall:

   Consist of four bones: maxillary, Lacrimal, ethmoid and sphenoid.

Thyroid eye diseases:

Soft tissue involvement:

- **Symptoms:**
  1. Grittiness
  2. Photophobia
  3. Lacrimation
  4. Retrobulbar discomfort
- **Signs:**
  1. Peri-orbital and lid swelling
  2. Conjunctival and episcleral hyperaemia
  3. Chemosis
  4. Superior limbic keratoconjunctivitis
  5. Keratoconjunctivitis sicca
- **Management:**
  1. Topical lubricants
  2. Head elevation with three pillows during sleep
  3. Taping of the eyelids during sleep

Lid retraction:

- **Signs:**

- ▪ Upper lid margin is either with or above the superior limbus
- ▪ Lower lid retraction suspected when sclera shows below the limbus.

1. Darymple: is lid retraction in primary gaze
2. Von Graefe: signifies retarded descent of the upper lid on down gaze
3. Kocher: a staring and frightened appearance of the eyes.

- **Management:**

1. Inferior rectus recession
2. Mullerotomy
3. Recession of the lower lid retractors

Proptosis:

- **Signs:**

Proptosis is axial, unilateral or bilateral, symmetrical or asymmetrical, and frequently permanent.

Sever Proptosis may compromise lid closure, with resultant exposure keratopathy and corneal ulceration.

- **Management:**

1. Systemic steroids:
   a. Oral Prednisolone
   b. Intravenous methylprednisolone
2. Radiotherapy
3. Combined therapy with irradiation, azathioprine and low-dose Prednisolone.
4. Surgical decompression

Optic neuropathy:

- **Symptoms:**

Impairment of central vision

- **Signs:**

1. Visual acuity is usually reduced
2. Visual field defect may be central or paracentral and may be combined with nerve fiber bundle defects.
3. The optic disc is usually normal, occasionally swollen and rarely atrophic.

- **Treatment:**

1. Intravenous methylprednisolone
2. Orbital decompression

Restrictive myopathy:

- **Signs:**

1. Ophthalmoplegia which may be permanent
2. Ocular motility is restricted initially by inflammatory edema and later by fibrosis
3. IOP may increase in up gaze
4. The four ocular motility defects are:
   a. Elevation
   b. Abduction
   c. Depression
   d. Adduction

- **Management:**

1. Surgery
2. Botulinum toxin injection into the involved muscle.

Infections:

Preseptal cellulitis:

Is an infection of the subcutaneous tissues anterior to the orbital septum.

• **Causes:**

1. Skin trauma
2. Spread of local infection
3. From remote infection of the upper respiratory tract or middle ear.

• **Signs:**

Unilateral, tender, and red peri-orbital and lid edema

• **Treatment:**

1. Oral co-amoxiclav
2. Intramuscular benzylpencillin
3. Oral flucloxacillin

Bacterial orbital cellulitis:

Is life-threatening infection of the soft tissues behind the orbital septum.

• **Causative organisms:**

1. Streptococcus pneumonia
2. Staphylococcus aureus
3. Streptococcus pyogenes
4. H. influenza

• **Symptoms:**

Rapid onset of sever malaise, fever, pain and visual impairment.

• **Signs:**

1. Unilateral, tender, warm and red peri-orbital and lid edema.
2. Proptosis (is most frequently lateral and downwards)

3. Painful Ophthalmoplegia

4. Optic nerve dysfunction

• **Management:**

1. Hospital admission

2. Antibiotic therapy

3. Optic nerve function should be monitored every 4 hours by testing papillary reactions, visual acuity, color vision and light brightness appreciation

4. Investigations:

   a. White cells count

   b. Blood culture

   c. CT of the orbit, sinuses and brain

5. Surgical intervention

Chapter Twelve
Ocular Trauma

Ocular Trauma

• Ocular Injury to the eye can result in:

  ▪ Immediate or delayed loss of vision.

  ▪ Significant disfigurement with great social and psychological impacts

• Recognition of the nature and extent of injury must be made prior to further damage from:

  ▪ Loss of ocular contents

  ▪ Infection

  ▪ Subsequent glaucoma or retinal detachment.

Assessment:

History:

▪ IT IS ESSENTIAL TO OBTAIN AN ACCURATE HISTORY FIRST Regarding:

  - Mechanism of injury: blunt, penetrating, chemical..etc

  - Time and place

  - Specific symptoms: loss of vision, diplopia

Examination:

ALWAYS BE GENTLE

▪ Look for asymmetry of the eyes

▪ Inspect of the eyelids

▪ Test best corrected VA

▪ Examine the pupillary reactions: look for afferent defect

▪ Measure IOP: look for soft eye or a hard eye

▪ Proceed with systemic examination of the anterior segment and posterior segment

ALWAYS REMEMBER

OCULAR SYMMETRY IS NORMAL

OCULAR ASYMMETRY IS ABNORMAL

MORE MISTAKES ARE MADE, I N MEDICINE

NOT LOOKING THAN BY NOT KNOWING

IF YOU DON'T KNOW . . . ASK!

- If you suspect a globe rupture at any point of the examination:
  - Stop
  - Protect eye
  - Call ophthalmology
  - NPO, Ancef IV
  - Make sure tetanus is up-to-date
  - CT orbits STAT
- RAPD present
  - Retinal detachment
    - extensive
  - Optic Nerve damage
    - Contusive
    - Laceration
    - RAPD not present, with diminished vision, consider:
  - Hyphema
  - Cataract
  - Vitreous hemorrhage

Types of Ocular Injury:

- Minor Ocular Trauma
- Chemical Injury
- Blunt Injury
- Orbital and Lid Injury
- Perforating with or without intraocular foreign body.

Minor Trauma

Conjunctival FB:

- SUBTARSAL FOREIGN BODY: No ocular examination is complete until the upper eyelid is everted and closely inspected.
- Linear epithelial defects is suggestive of a foreign body under the eye lid

Corneal FB:

- Corneal foreign bodies and rust rings are best removed with a sterile disposable needle (19 to 23 G).
- Do not attempt central or deep foreign bodies as a slit lamp is required to avoid excessive trauma.
- Instil antibiotic ointment and pad for 24 hours.
- Review daily until healed, or referred to ophthalmologist.
  - Corneal abrasions and flash burns (from UV burns) can cause severe pain which is self limiting as the cornea heals.
  - Can use local anaesthetic drops for examination and fluorescein staining.
  - Always evert the upper lid to check for a subtarsal foreign body.

DO NOT ALLOW PATIENT TO USE LOCAL ANAESTHETIC DROPS.

  - While the cornea will usually heal in 24 hours with a firm doubled pad,
  - healing will be delayed by the use of local anaesthetic drops and severe ulceration can occur.

CHEMICAL INJURY

- Alkali burns are twice as common as acid burns since alkalis are more widely used at home and in industry.
- The most common involved alkalis are ammonia, sodium hydroxide and lime.

- The commonest acids implicated are sulphuric, sulphurous, hydrofluoric, acetic, chromic and hydrochloric.
- Acid will coagulate proteins but alkali will continue to denature the protein of the cornea.
- A chemical burn is the only type of eye injury that requires immediate treatment without first taking a history and performing a careful examination. Immediate treatment is as follows:
  - Irrigation is crucially important in minimizing of contact of the chemical with the eye and duration the pH as soon as possible. Normal saline normalizing (or equivalent) should be used to irrigate the eye for 15- 30 minutes or until pH is normalized.
  - The eyelids should be double- everted so that any retained particulate matter trapped in the fornices, such as lime or cement, is removed.
  - Necrotic areas of corneal epithelium should be to allow for proper reepithelialization. debrided
- Mild injuries are treated with a short course of topical steroids, cycloplegics and prophylactic antibiotics for about 7 days.
- The main aims of treatment of more severe burns are to reduce inflammation, promote recovery and prevent sterile corneal ulceration. epithelial

BLUNT INJURY

- Blunt injury is common. often caused by fist or squash ball.
- May be hidden initially by lid swelling and 'black eye'.
- Intraocular haemorrhage is manifest by:

HYPHAEMA

- which may appear as a blood level or fill the entire anterior chamber.
- The more severe injury may also have retinal haemorrhage.
- Later complications include retinal detachment and secondary glaucoma.

- May rebleed in first week with subsequent glaucoma and corneal blood staining. Surgical removal of the clot may be required.
- SEVERE HYPHAEMA will need hospitalisation with bed rest for five days to lessen risk of rebleed. Single eye pad preferred. AVOID USE OF ASPIRIN.

RETINAL DETACHMENT.

- Beware of floaters, flashes and field defects.
- Urgent referral is mandatory.
- Rupture of the globe may result from very severe blunt trauma.
- The rupture is usually anterior, at or near the limbus with prolapse of uveal tissue.
- Occasionally, the rupture is posterior (occult) with little damage to the anterior segment.
- Clinically, occult rupture should be suspected if there is of anterior chamber depth and intraocular asymmetry pressure in the affected eye is low.

CORNEAL LACERATION

- Comeal laceration is recognised by:
    - Iris prolapse with pupil distortion and iris bulging through cornea.
    - Shallow or flat anterior chamber. Compare distance between the cornea and iris plane with the other eye.
    - May have associated blood level in the anterior chambre:(HYPHAEMA).
- Extension of the laceration into the SCLERA increase the risk of damage to lens and retina.
- Loss of vitreous gel via a posterior injury may be evident. Often associated vitreous haemorrhage results in loss of red reflex and poor vision.
- Lacerations through the eyelids can result in scleral lacerations without associated corneal lacerations.
- The loss of pressure wirhln the eye may resultin the eyewall to collapse and lose its spherical shape

Penetrating trauma

- Penetrating injuries are more common in males than females by a 3: 1 ratio, and in the younger age group.
- The most common causes are assault, domestic accidents and sport.
- The extent of the injury is determined by the size of the object, its speed at the time of impact and its composition.
- Sharp objects such as knives will cause a well-defined laceration of the globe.
- However, the extent of damage caused by flying foreign bodies is determined by their kinetic energy. For example, an airgun pellet is large and although relatively slow moving has a high kinetic energy
- In contrast, a fast moving fragment of shrapnel has a low mass, and therefore will cause a well- defined laceration and less intraocular damage than an air gun pellet.
- Tractional retinal detachment following penetrating trauma is the result of vitreous incarceration in the wound and the presence of blood within the vitreous gel which acts as a stimulus to fibroblastic proliferation along the planes of incarcerated vitreous.
- The contraction of such membranes leads to shortening and a effect on the peripheral retina in the region of rolling the vitreous base and eventually to tractional retinal detachment.
- If suspected, lightly pad eye and arrange for urgent ophthalmic assessment. Will need good illumination and magnification for complete examination.

Prognosis:

- Prognosis of ocular injuries is determined by the following:
  - Infection
  - Extent of damage to the retina
  - Damage to the lens
  - Corneal scarring

- Intraocular foreign bodies
- Sympathetic ophthalmia:

• Is an uncommon devastating condition whereby the **OTHER EYE** responds with often a severe inflammatory reaction and loss of vision following an injury involving the uveal tissue within the eye.

• With severe eye injuries where there is no prognosis for

visual recovery the injured eye may be removed to lessen the risk of this condition.

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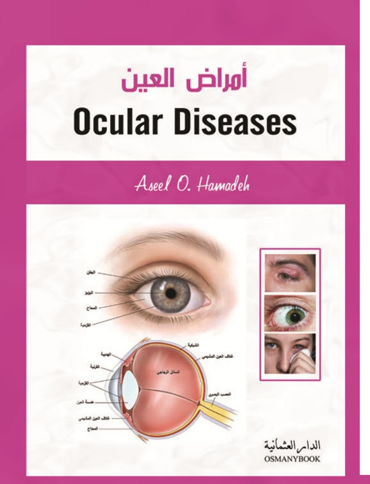
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Ocular Diseases

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